APOLLO HOSPITALS,

SECUNDERABAD

ICU MANUAL

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QUALITY DEPARTMENT

1. ICU ADMISSION, DISCHARGE AND TRIAGE

GICU

ADMISSION CRITERIA

Patient are admitted to the GICU for evaluation and treatment of illnesses that may lead to death or acute organ failure, that are amenable to treatment, and that require intensive monitoring (including intravascular pressure monitors), frequent evaluation (including laboratory tests), continuous medication infusions, and/or intensive nursing care. Admission decisions are made based on complete evaluation of the patient's medical and nursing requirements. Besides, all patients from General Surgery, ENT, Head and Neck Surgery, Urology, Surgical Gastroenterology, Neurosurgery, Plastic surgery and Orthopedic Surgery for whom ICU care is requested. Adult Patients are provided admission to this unit. In case of shortage of beds in other ICUs, patients of other specialties' may also be admitted.

A. Respiratory System

1. Acute respiratory failure
2. Chronic respiratory failure with altered sensorium and Haemodynamics.
3. Ventilation or oxygenation dependent on patient condition.
4. Require tracheal suctioning or chest physiotherapy every 2 hours or more often.
5. Acute or incipient airway obstruction.

B. Cardiovascular system

1. Severe hypertension or hypotension.
2. Severe Bradycardia or tachycardia with hemodynamic instability.

C. Gastrointestinal system

1. Acute upper or lower GI tract hemorrhage causing orthostatic hypotension or blood loss requiring multiple transfusions.
2. Hepatic dysfunction causing encephalopathy.
3. Inflammatory bowel disease causing ileus or peritonitis.

D. Renal system

1. Persistent oliguria.
2. Myoglobinuria.

E. Endocrine system

1. Diabetic emergencies – DKA/HONK/Hypoglycemia with loss of Consciousness.
2. Severe acidosis.
3. Severe electrolyte abnormalities.
4. Thyroid emergencies.

F. Hematology system

1. Thrombocytopenia with active bleeding.
2. Coagulopathy with active bleeding.

G. Central nervous system

1. Abnormal Glassgow coma score with need for continuous monitoring.
2. Uncontrolled seizures.
3. Progressive muscle weakness involving respiratory muscles.

H. Infectious/Environmental agents

1. Severe Sepsis / Septic shock.
2. Acute meningitis with neurological abnormalities.
3. Acute poisoning with suppressed level of consciousness or airway compromised, hemodynamic instability or incipient cardiac arrhythmias.
4. Acute drug withdrawal syndromes with hemodynamic instability.

DISCHARGE CRITERIA

Patients are discharged from the GICU when their acute illness is treated, their medical condition has stabilized, and they do not require frequent evaluation (including laboratory tests), continuous medication infusions, and/or intensive nursing care. Discharge decisions are made based on complete evaluation of the patient's medical and nursing requirements.

A. Respiratory system

1. Treatment and reversal of respiratory failure with stable haemodynamics.

B. Cardiovascular system

1. Blood pressure within acceptable range sans ionotropic support.
2. Symptomatic Bradycardia or tachycardia reversed.
3. Not requiring continuous infusions or vasoactive drugs or antiarrhythmic Agents.
4. No pulmonary artery catheters.
5. Pericardial tamponade resolved for > 24 hours.

C. Gastrointestinal system

1. No orthostatic hypotension and hematocrit stable for previous 24 hours.
2. Stable or improving hepatic encephalopathy, if present during current Admission.
3. Ileus or peritonitis resolving, if present during current admission.

D. Renal system

1. Oliguria corrected or dialysis program established.

E. Endocrine system

1. Correction of diabetic emergencies with treatment stabilizing blood sugars for 24 hrs.

F. Hematology system

1. Control of bleeding and correction of Thrombocytopenia.

2. Correction of INR with control of bleeding.
3. Stable hematocrit for 24 hrs.

G. Central nervous system

1. Improving Glassgow coma score with no need of continuous monitoring.
2. Seizures controlled on stable medical regimen.

H. Infectious/Environmental agents

1. Resolving Sepsis with acceptable blood pressure
2. Stable or improving neurological function with resolving acute meningitis.
3. Stable or improving consciousness and airway protection with resolving acute poisoning, without need for further monitoring.

CRITERIA FOR ADMISSION OF SURGICAL PATIENTS:

1. Patient in hypotension with sepsis.
2. Peri-operative acute respiratory failure.
3. Treatment of hemodynamic instability status.
4. Intubated patient requiring ventilator support.
5. Severe tachycardia or bradycardia.
6. Respiratory distress.
7. Persistent oliguria.
8. Acute postoperative patients requiring intensive monitoring of airway, selected perfusion, respiratory status, or mental status
9. Acute postoperative patients requiring continual acute monitoring and manipulation of intravenous fluid therapy

DISCHARGE CRITERIA FOR SURGICAL PATIENTS:

The following will include absolute criteria necessary for discharge and relative physiologic criteria to be used in guidelines for discharge.

Criteria for Discharge from ICU care.

Transfer / discharge will be based on the following criteria:

1. Stable hemodynamic parameters.
2. Stable respiratory status (patient extubated with stable arterial blood gases) and airway patency.
3. Minimal oxygen requirements that do not exceed patient care unit guidelines.
4. Intravenous inotropic support, vasodilators, and antiarrhythmic drugs are no longer required.
5. Cardiac dysrhythmias are controlled.
6. Patients with mature artificial airways (tracheostomies) who no longer require excessive suctioning.

CT POST ICU

ADMISSION CRITERIA

1. Adult post operative patients after cardio-thoracic and vascular surgery.
2. Patients after cardiac / cardio-thoracic surgery having:

    i. Acute hypotension.
    ii. Life threatening arrhythmias.
    iii. Acute respiratory failure with cardiovascular instability.
    iv. Hemodynamic instability requiring continuous monitoring and/ or constant infusion of vasoactive drugs.
    v. Endotracheal intubation and mechanical ventilation.

DISCHARGE CRITERIA from CT Post ICU.

1. Correction of hypotension.
2. Control/correction of life-threatening arrhythmia.
3. Correction of respiratory failure.
4. Hemodynamic stability.
5. Not dependent on mechanical ventilator.

Transfer/discharge will be based on the following criteria:

1. Stable haemodynamic parameters.
2. Stable respiratory status (patient extubated with stable arterial blood gases) and airway patency.
3. Minimal oxygen requirements that do not exceed patient care unit

Guidelines.
4. Intravenous inotropic support, vasodilators, and antiarrhythmic drugs are no longer required.
5. Cardiac dysrhythmias are controlled.
6. Removal of all hemodynamic monitoring catheters.
7. Discontinuation of peritoneal or hemodialysis with resolution of critical illness.
8. Patients with mature artificial airways (tracheostomies) who no longer require excessive suctioning.

CICU

Patients from Cardiology services are provided admission to this unit. In case of bed shortage, other specialty patient may also be admitted.

ADMISSION CRITERIA

Cardiovascular system

1. Severe hypotension or hypertension.
2. Severe Bradycardia or tachycardia with haemodynamic compromise.
3. Acute myocardial infarction.
4. Life threatening dysrhthmia.
5. Continuous infusion of vasoactive drugs.
6. Continuous infusion of antiarrhythmic drugs.
7. Pericardial effusion with tamponade.
8. Post angiography / angioplasty patients needing continuous monitoring.
9. Patients with cardiogenic shock.

DISCHARGE CRITERIA

Cardiovascular system

1. Correction of hypotension / hypertension.
2. Correction of Bradycardia / Tachycardia.
3. Absence of life-threatening arrhythmia after observation.
4. Not requiring continuous infusions of vasoactive drugs or antiarrhythmic

Agents.
5. Pericardial tamponade resolved > 24 hrs.
6. Haemodynamic stability.

Transfer / discharge will be based on the following criteria:

1. Stable haemodynamic parameters.
2. Stable respiratory status (patient extubated with stable arterial blood gases) and airway patency.
3. Minimal oxygen requirements that do not exceed care unit guidelines.

4. Intravenous inotropic support, vasodilators, and antiarrhythmic drugs are no longer required.
5. Cardiac dysrhythmias are controlled.
6. Discontinuation of peritoneal or hemodialysis with resolution of critical Illness.
7. Patient with mature artificial airways (tracheostomies) who no longer require excessive suctioning.

TRIAGE OF ICU

Under ideal conditions patients shall be admitted or discharged strictly on their potential to benefit from ICU care. Unfortunately, in many instances the number of potential ICU patients exceeds the available beds. A method of prioritizing or triaging patients is necessary. Initial triage or patients may follow the guidelines given in the prioritization model. In an environment where ICU admissions are rigorously screened for benefit and discharge is ongoing and continuous, the need for triage is minimized. Triage decisions are made explicitly, and without bias. Ethnic origin, race, sex social status, sexual preference or financial status is never considered in triage decisions.

Prioritization Model

This system defines those that shall benefit most from ICU (Priority 1) to those that shall not benefit at all (Priority 4) from ICU admission.

Priority 1: These are critically ill, unstable patients in need of intensive treatment and monitoring that cannot be provided outside of the ICU. Usually, these treatments include ventilator support, continuous vasoactive drugs infusions, etc. Priority 1 patients generally have no limits placed on the extent of therapy they are to receive. Examples of these patients may include post-operative or acute respiratory failure patients requiring mechanical ventilatory support and shock or hemodynamically unstable patients receiving invasive monitoring and / or vasoactive drugs.

Priority 2: These patients require intensive monitoring and may potentially need immediate invertension. No therapeutic limits are general stipulated for these patients. Examples include patients with chronic comorbid conditions who develop acute severe medical or surgical illness.

Priority 3: These unstable patients are critically ill but have a reduced likelihood of recovery because of underlying disease or nature or their acute illness. Priority 3 patients may receive intensive treatment to relieve acute illness but limits on therapeutic efforts may be set.

Priority 4: These are patients who are generally not appropriate for ICU admission. Admission of these patients shall be on an individual basis, under unusual circumstances. These patients can be placed in the following categories:

Little or no anticipated benefit from ICU care based on low risk of active intervention that could not safely be administered in a non-ICU setting (too well to benefit from ICU care). Examples include patients with peripheral vascular surgery; hemodynamically stable diabetic ketoacidosis, mild congestive heart failures, conscious drug overdose, etc.

Patients with terminal and irreversible illness facing imminent death (too sick to benefit from ICU care). For example: severe irreversible multi-organ system failure, metastatic cancer unresponsive to chemotherapy and / or radiation therapy (unless the patient is on a specific treatment protocol), patients with decision-making capacity who decline intensive care and / or invasive monitoring and who receive comfort care only, brain dead non-organ donors, patients in persistent vegetative state, patients who are permanently unconscious, etc.

PERFORMANCE REVIEW

The performance evaluation and review of an ICU shall include its admission/discharge/triage policy. A multi-professional team shall review performance at least annually. In order to adequately review performance as it relates to admission, outcome, and the decision-making process.

Administrative Recommendations to Facilitate Appropriate Admissions, Discharges and Delivery of Intensive Care Units

The ICU Co-ordinator is appointed on the basis of training, interest, type of practice, and availability and who can give clinical, administrative and educational direction to the Intensive Care Unit.

The ICU Coordinator shall assume responsibility for assuring the quality, safety, and appropriateness of care in the Intensive Care Unit.

The ICU Coordinator shall work collaboratively with the Coordinators of other areas in the institution so that patent care, triage, and patient flow are effective and efficient.

BED SHORTAGE POLICY

SUBJECT
Contingency plan for handling a shortage of Intensive Care Beds in Intensive Care Units.

PURPOSE
This policy issuance sets forth a contingency plan for handling a shortage of intensive care beds in any of the ICUs.

POLICY

1. When no bed is available in a given ICU for a patient needing admission to that ICU, and when, in the opinion of the consultant, no patient presently occupying a bed in that ICU can be moved to a general patients care unit, physicians in that ICU may seek to locate a bed in the other ICU.

2. If a bed is available in an alternate unit, the patient may be admitted to that unit only with the consent of its Consultant. Responsibility for medical care of the patient shall rest with the staff of the unit of which the patient has been admitted.

3. Consultants of the two ICUs agree to allow other units' Consultant to admit and care for a critically ill patient in their unit, when shortages as outlined here arise, unless there are medical contraindications. Aside from those exceptions, one ICU group shall be allowed to "borrow" an ICU bed in another unit with the understanding that this represents a temporary

admission, and that the patient shall promptly be returned to the appropriate ICU as soon as a bed is available.

4. Because a bed shortage may occur at any hour of the day or night, each ICU coordinator shall have ultimate responsibility for deciding which patient, if any, may be moved from that ICU to make room for a case more urgently in need of specialized care.

INFECTION CONTROL IN ICU

A. Prevention and containment of Hospital Associated Infections (HAI) / Nosocomial infection is a fundamental principle of effective medical practice.

B. The critically ill patient is highly vulnerable to nosocomial infection, which results in significant morbidity, prolonged length of hospital stay, increased cost and attributes mortality.

C. It is the responsibility of every member of the health care team to ensure compliance with hospital and unit infection control policies.

D. This may include reminding senior colleagues or visiting teams to conform to basic issues such as hand washing or barrier nursing measures.

E. Hand-washing remains the only established method of effective infection control and shall be religiously performed by all members or the health care team.

1. Compulsory before and after entering or the health care team.

- Physical examination of the patient.
- Manipulation of patient's environment including respiratory equipment, infusion pumps, dressings, drains, linen or bedding.
- Inspection or handling of the patient chart, case notes etc. when these are placed inside the cubicle.
- Following all procedures, even if aseptic techniques are used.

2. This may be performed by washing hands using AHD hand cleanser for minimum (at the basin closest to, or within the patient's cubicle) (or by soap and water or by alcohol based hand rubs, for example AHD 2000/sterilium).

E. Gloves

1. Disposable gloves shall be worn for all contacts with patient's body fluids, dressings and wounds.

2. The use of gloves is not a substitute for hand washing before and after patient contact..

3. Gloves shall be disposed of in appropriate waste disposal containers.

F. Barrier nursing measures:

The following patients are regarded as infective risks requiring barrier nursing:

1. Infection or colonization with
   (a) Methicillin Resistant Staph. Aureus.
   (b) Vancomycin Resistant Enterococcus.
   (c) Multiresistant Gram Negatives.
   (d) Toxin A Producing Clostridium Difficile.

2. Febrile Neutropenia.

3. High risk immunosuppressed patients as directed by Infection Control Team.

G. Aseptic Technique:

1. Aseptic technique is to be used for all patients undergoing major invasive

procedures.
This includes :

a) Hand disinfection surgical scrub.
b) Sterile barrier: full gown, mask, disposable caps, gloves and sterile drapes .
c) Skin preparation with Povidone Iodine for example, Betadine: let the skin dry.
d) Proper sharps disposal.
e) The person performing the procedure is responsible for disposal of all sharps (needles, blades) using the sharp disposal containers.
f) Nursing staff is not responsible for cleaning-up sharps after a medical procedure.

H. "Traffic Control"

1. Movement of people through the Unit shall be kept to a minimum. This applies equally to visiting clinicians and large numbers of relatives.

2. All visitors are expected to conform to the above infection control measures and are to be tactfully reminded or instructed about these issues.

3. All the visitors are advised to wash their hands before entering & leaving the ICU.

GUIDELINES TO MEDICAL STAFF.

A. Admitting a new patient

1) Obtain as much information as possible from relatives and referring physicians.

2) Primary survey

a. Ensure adequate airway, breathing and place patient on highest FiO_2.

b. Check circulation and venous access.

3) Secondary survey (fully examine patient)

a. Document essential orders:

i. Ventilation.

ii. Sedation / analgesia.

iii. Drugs, Infusions.

iv. Fluids.

4) Outline plan to nursing staff.

5) Secure appropriate basic monitoring / procedures:

a. SpO2.

b. ECG.

c. Arterial line, if needed.

d. Nasogastric tube.

e. CVP monitoring if needed.

6) Basic investigations:

a. Routine biochemistry, blood picture and coagulation studies.

b. Microbiology as indicated.

c. Arterial blood gas as indicated.

d. CXR (after performing appropriate Hemogram).

7) Assess the need for advanced investigations CT, angiography, MRI, etc.

8) Advanced monitoring where indicated for example, PA catheter, ICP, SpO2,

9) Document the details in case notes.

10) Inform and counsel relatives.

B. Daily management
Daily investigations:

a. Routine blood tests (Biochemistry and Hematology) are ordered on the physician Order Sheet. Drug levels or other tests are requested as required.
b. The junior medical staff is responsible for taking blood samples when the nursing staff request assistance.
c. Chest x-rays are ordered.
d. Handover ICU rounds are done. These are brief business rounds to handover essential information to the next team (either day or night) and are attended by the unit residents, registrars and nurses.
e. Complex investigations (for example, CT, MRI scans) and procedures shall be authorized by the Senior Consultants / ICU in-charges.

C. Documentation

The following guidelines are designed to facilitate the recording of clear, relevant information that is essential for continuity of care, audit and medico legal review. Entries shall establish a balance, being concise but still accurately recording all relevant information and events.

1. Documentation by ICU registrars/consultants includes:

a. Admission note for all patients admitted to ICU.

b. Daily entry in case notes.
c. Discharge summary.
d. Death Certificate.
e. In-house Transfer Summary.
f. Physical Restraint Form.
g. Consent form.

2. ICU Admission Note

a. All patients admitted to the ICU shall have an admission noted.
b. The Consultant shall be notified and invited to record an admission note.
c. The admission note shall incorporate all relevant aspects of the patent's medical history, clinical examination and results of appropriate investigations.

3. Daily Progress Note in ICU

a) A daily entry shall be made in the case notes:

Notes shall mention plan of care, pain assessment, effect of medication, patient and family education.

b) Additional notes shall be made for the following:

1. Significant changes in physical condition necessitating changes in management, for example, renal failure requiring dialysis.
2. Major procedures, for example, laparotomy, tracheostomy, etc.
3. Results of specific investigations or test, for example, CT scans, endocrine tests, etc.

4. In-House Transfer Forms

a. All patients transferred out of ICU require an In-House Transfer form to be completed.

b. This is single page document outlining all relevant transfer information.

c. The reverse side of the form is a nursing transfer summary, which shall be completed by the attending nurse.

d. The consultant on duty on day of transfer is responsible for completing the form.

5. Consent in ICU

a. Competent patients:

1. All competent patients undergoing invasive procedure in ICU shall have consent for procedure completed and signed by the patient.

b. Incompetent patients (sedation, coma or encephalopathy):

1. Third party consent is necessary for routine ICU procedures, these include:

(a) Endotracheal tubes.
(b) Arterial lines.
(c) Central venous lines.
(d) Pulmonary artery catheters.
(e) Transvenous pacing wires.

(f) Underwater seal drains.
(g) Intra-aortic balloon counterpulsation.
(h) Bronchoscopy.

2. Ultimate responsibility for consent lies with the operator performing the procedure, however junior medical staff shall ensure appropriate consent is obtained.

3. Relatives shall always be informed of any procedures and the consent issue explained.

D. ICU Round

1. The daily ICU round is an integral feature of the running of the Unit. It is the forum to openly discuss management issues and is a useful teaching forum

2. ICU incharge on duty is expected to present their allocated patients during this round and actively participate in the discussion. Presentations during this round shall be of a standard suitable for a fellowship examination.

3. The ICU round is attended by

a. ICU consultants, specialists.
b. Nursing staff.

4. Presentation at ICU round:

Presentation shall take no more than 5-10 minutes.
Emphasize the relevant and pertinent issues only.

a. Patient details and demographics.
b. Diagnosis or major problems.
c. Relevant pre-morbid history pertinent to this admission.
d. Relevant progress and events in ICU (deterioration/ improvement, procedures, investigations).

e. Current clinical status (system by system).
f. Outline features on daily basis for Pathology and Radiology.
g. Current plan of management.
h. Medications.
i. Further investigations/ procedures.
j. Discharge / Prognosis.

5. Laboratory results:

Biochemistry, hematology and coagulation results for the ICU round shall be obtained by the nurse responsible for the patient, either by phone or via the computer terminals in each nursing station.

E. Hospital Emergencies.

1. In case of any emergency, inform the concerned authorities, state nature and location of emergency. (Following the Safety Manual / the Red Book).

2. Fire (follow the Safety Manual).

a. A copy of the hospital Safety Manual (fire, smoke, and bomb-threat) is kept in all nursing stations.

b. The Fire Officer is the overall controller during a fire or smoke emergency (Code Brown).

c. Become familiar with the location of fire exits, extinguishers in the unit.

i. Unless a fire is small and easily contained do not attempt to fight alerting other staff members as indicated.

ii. Remove yourself from the immediate vicinity of the fire, alerting other staff members as indicated.

iii. Wait for the arrival of the Fire Officer and assist in any patient movement/ evacuation only as indicated by the Fire Officer.

iv. In the event of a significant fire / smoke hazard, staff shall only re-enter the danger zone in the immediate company of a fire fighter, with appropriate breathing apparatus.

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6. CLINICAL PROCEDURES

A. Introduction

1. Invasive procedures shall only be authorized by the Consultant/ ICU Incharge.
2. Adequate familiarization and supervision with unfamiliar procedures is essential, there is always someone available to help.
3. The relative risk vs. benefit of all procedures shall be carefully considered.
4. Do not persist if you are having difficulty with the procedure: call for help.
5. Consent for procedures:
   a. Competent patients undergoing invasive procedures shall have the Consent Form completed and signed by the patient.
   b. Third party consent is necessary for incompetent patients undergoing routine ICU procedures.
6. Indications, conduct and any complications of the procedure shall be clearly documented in the case notes in addition to a consent form is completed.
7. Discuss the planned procedure with the ICU nursing staff and allow sufficient time for setting up of trays and equipment.
8. It is the responsibility of the operator to discard all sharps used in the procedure and to ensure that they are placed in a sharps disposal container as per protocol.

B. Procedures.

1. Specialized procedures are done by the Consultant.
2. Protocols for the under mentioned routine and specialized are outlined in the following sections:

Routine ICU Procedures:

1. Endotracheal intubation.
2. Central Venous Catheterization.
3. Arterial cannulation.
4. Urinary Catheterization.
5. Lumbar puncture.
6. Pleurocentesis.
7. Peritoneocentesis.
8. Nasogastric tube insertion.

Specialized ICU procedures:

1. Fiber-optic bronchoscopy.
2. Transvenous pacing.
3. Pericardiocentesis.
4. Esophageal tamponade tube insertion.
5. Intra-aortic balloon counterpulsation.
6. Epidural catheterization.
7. Underwater seal drain insertion.

A. Peripheral Catheters:

1. Indications

a. First line IV access for resuscitation including blood transfusion.
b. Stable ICU patients where a CVC is no longer necessary.
c. Arterial Catheters.

2. Management Protocol:

a. Remove all resuscitation lines inserted in unsterile conditions as soon as

possible.

b. Brachial and Femoral arterial lines shall be changed as soon as Radial or Dorsalis Pedis arteries are available.
c. Aseptic technique:
   i. Hand wash and gloves.
   ii. Skin preparation with Betadine.
d. Local anesthesia in conscious patients.
e. Cannulae:
   i. Arrow (Seldinger Technique): Radial or Femoral kits.
   ii. Single lumen 18G CVC for Femoral arterial lines.
f. Sites: (in order of preference): Radial, Dorsalis Pedis, Ulnar, Brachial, Femoral.
g. The Femoral artery may be the sole option in the acutely shocked patient.
h. Dressing: Occlusive Opsite ® (Sutured)/Tegaderm/ Centragard transparent dressing.
i. There is no optimal time for an arterial line to be removed or changed.
j. IA cannula is changed / removed only in the following settings.
   i. Distal ischemia.
   ii. Mechanical failure (over damped waveform, inability to aspirate blood).
   iii. Evidence of unexplained system or local infection (of CVC lines).

k. Measurement of pressure:
   i. Transducers shall be 'zeroed' to the mid-axillary line
1. Maintenance of lumen patency:
   i. Continuous pressurized (intraflo ®) Heparinised Saline flush (lu/ml) at 3 ml/hr.

3. Complications.

a. Infection.
b. Thrombosis.

c. Digital ischemia.
d. Vessel damage / aneurysm.
e. HITS (secondary to Heparin infusion).

B. Central Venous Catheters.

Note : All doctors shall be familiar with the interpretation and limitation of haemodynamic variables derived from central catheters (CVC and PAC) in critically ill patients

1. Indications:

a. Standard IV access in ICU patients:
    i. Fluid administration (including elective transfusion).
    ii. TPN, hypertonic solutions (Amiodarone, Nimodepine, etc).
    iii. Vasoactive infusions.
b. Monitoring of right atrial pressure (CVP).
c. Venous access for:
    i. Pulmonary artery catheterization.
    ii. Continuous replacement therapy (CVVHDF), plasmapheresis.
    iii. Jugular bulb oximetry.
    iv. Transvenous pacing.

d. Resuscitation:
    i. Done to large bore peripheral IV or large bore central venous catheter standard 3-lumen CVCs are not appropriate for acute volume resuscitation (consider a PAC sheath).

2. Management protocol.
a. Sites

i. Subclavian is the preferred site for routine stable patients, followed by Internal Jugular.

ii. Femoral access is preferable where:

(a) Limited IV access (burns, multiple previous CVC's).

(b) Thoracic approach is considered hazardous:
(1) Severe respiratory failure from any cause ($PaO_2/FiO_2<150$).
(2) Hyperexpanded lung fields (severe asthma, bullous lung disease).
(3) Coagulopathy (see below).

b. Coagulopathic patients:

i. INR more than 2.0 or APTT more than 50 seconds, correct with FFP.
ii. Platelets less than 50,000, transfuse platelets.
iii. Failure to increment platelet count after transfusion (avoid subclavian).
iv. Uncontrolled Coagulopathy (use femoral approach).

c. Technique policy:

i. Local anesthesia in awake patients.
ii. Strict aseptic technique at insertion.

(a) Hand disinfection: Surgical scrub with Chlorhexidine for more than 1 Minute.
(b) Sterile barrier: gown, glove, cap and mask and sterile drapes.
(c) Skin preparation.

iii. Seldinger technique only.
iv. Suturing all lines.
v. Dressing non-occlusive dressing.
vi. Flush all lumens with Heparinised Saline.

d. Maintenance:

i. Routine IV administration set change every day.
ii. Daily inspection of the insertion site and clinical examination for infection irrespective of duration of insertion.
iii. Catheters are left place as long as clinically indicated and changed when.

(a) Evidence of systemic infection:
(1) New, unexplained fever.
(2) Unexplained rise in WBC.
(3) Deterioration in organ function.
(4) Positive blood culture by venipuncture with likely organisms (S. Epidermidis, Candida Sp.).

(b) Evidence of local infection-inflammation or pus at the insertion site:

iv. Guide wire exchanges are actively discourage. They may be indicated in the following situations, after discussion with a Consultant.

(a) Mechanical problems in a new catheter (leaks or kinks).

(b) Difficult or limited central access (for examples, burns).

v. Maintenance of lumen patency.

(1) Withdraw 2 ml and discard.
(2) Flush with 2 ml and discard.
(3) Flush 1.5 ml solution (5000 U Heparin / 2 ml).

3. Complications:

a. At insertion:

i. Arterial Puncture.

(a) Haematoma with mass effect.
(b) Arterial thrombosis / embolism.

ii. Pneumothroax, Haemothorax, Chylothorax.
iii. Neural injury (Phernic, Brachial Plexus, Femoral Nerve).

b. Passage of wire/ catheter:

i. Arrhythmias.
ii. Perforation of SVC, RA, tamponade.

c. Presence of catheter:

(a) Size of catheter – thicker catheters (Pulmonary Artery catheters, Vascaths).
(b) Site of catheter – Femoral more often than Internal Jugular, more often than subclavian sites.
(c) Number of lumens.
(d) Nature of fluid through catheters – TPN of Dextrose solutions.
ii. Thrombosis.
iii. Catheter / Air embolism.
iv. Knotting of catheters (especially PAC).
v. Pulmonary infarct / arterial rupture (PAC).
vi. HITS secondary to Heparin.

C. Urinary catheters

1. Standard in all ICU patients.

2. Management protocol:

a. Aseptic technique at insertion:
i. Hand disinfection: AHD for more than 1 minute.
ii. Sterile barrier: gloves and sterile drapes.
iii. Skin preparation: Chlorhexidine 1% Povidine iodine.
b. Local Anaesthesia gel in all patients.
c. Foley catheter for 7 days and changes to Silastic thereafter if prolonged

catheterization is anticipated. (i.e. more than 14 days).
d. Remove catheters in anuric patients and perform intermittent catheterization weekly, or as indicated.

D. Epidural Catheters.

1. Indications:
   a. Post operative pain relief (usually placed in theatre).

   b. Analgesia in chest trauma.

2. Management Protocol.

   a. Notify the Anesthesia of any epidural placed in ICU.
   b. Epidural cocktails shall follow the Anesthetic protocols.
   c. Strict aseptic technique at insertion.
   d. Daily inspection of the insertion site. The catheter shall not be routinely redressed, except under the advice of the Anesthetist.
   e. Leave in for a maximum of 5 days and then remove.
   f. Remove if not in use for more than 24 hours or clinical evidence of unexplained sepsis or positive blood culture by venipunture with likely organisms (S.Epidermidis, Candida).

3. Complications.

   a. Hypotension from sympathetic blockade / relative hypovolaemia.
      i. This usually responds to adequate intravascular volume replacement.
   b. Pruritis, nausea and vomiting, or urinary retention (Opioid effects).
   c. Post-dural puncture headache.
   d. Infection epidural abscess.
   e. Pneumothorax (rarely).

E. Pulmonary artery catheters.

1. Policy.

a. Insertion of PA catheters shall be authorized by the Senior Consultant.
b. Become familiar with the theory of insertion, indications, interpretation and complications of PACs.
c. Insertion of PA catheters shall never delay resuscitation of shocked patients.

d. Allow sufficient time for nursing staff to set up insertion trays and transducer manifolds.
e. Remove catheters once they are not being routinely used.

2. Indications.

a. Haemodynamic measurement (Cardiac output, stroke volume, SVR).
i. Diagnostic assessment of shock states (Cardiogenic, Hyperdynamic, Hypovolaemic).
ii. Assessment of response to treatment in the above.
b. Measurement of right heart pressure (RAP, PAP).
i. Acute pulmonary hypertension
ii. Pulmonary Embolism
iii. Cardiac tamponade
c. Estimation of preload / left heart filling (PAOP).
i. Intravascular volume status
ii. LVF
iii. Response to fluid loading
d. Measurement of intracardiac shunt ; (Acute VSD).
e. Derivation of oxygen variables (VO2, DO2) not routinely done in this unit

3. Management protocol.

a. Insertion protocol as per CVC.
b. Specific features of PACs.
i. Insertion protocol.
(a) Sheath introducer (8.5 Fr) with side port, haemostatic valve and plastic contamination shield.
(b) Shared transducer for RAP (Proximal) and PAP (Distal) lumens.
(c) Check competence of balloon and concentric position.

(d) Ensure all lumens are flushed with Heparinised Saline prior to Insertion.

(e) Ensure adequately zeroed system and appropriate scale (0-40 mmHg) on monitor prior to insertion.
(f) Insert catheter using changing waveforms (RA RV PA) on monitor with balloon inflated and locked until catheter displays Pulmonary Artery occlusion tracing: usually 50 cm catheter in most patients using Subclavian and left Internal jugular approach; right Internal jugular 40 cm.
(g) Deflate balloon and ensure adequate PAP trace. Adjust catheter depth until a PAOP trace appears with 1-1.5 ml air in balloon.
(h) Suture introducer and attach contamination shield to the hub of the Introducer.
(1) Apply a non-occlusive dressing.

ii. Ensure an adequate PA tracing is on the monitor at all times."wedged" tracings shall be corrected as soon as possible

(a) Flush distal lumen with 2ml normal saline
(b) Withdraw the catheter until a PA trace is visible

iii. Measurement of pressures

a. Reference pressures to the mid axillary line
b. Measure at end-expiration of the respiratory cycle

c. Do not disconnect ventilated patients to measure pressures
d. Measurement of PAOP

- End expiration lowest point in ventilated patients, highest point in spontaneously ventilating patients
- Use the "electronic cursor" on Marquette monitors after 2-3 respiratory cycles.

iv. Haemodynamic measurements

(a) These are routinely performed by the nursing staff, however junior medical staff shall be familiar with the procedure
(b) Record all measurements in the flow chare in the results folder
(c) Cardiac outputs

- Inject: 10 ml 5% Dextrose at room temperature
- Inject throughout the respiratory cycle
- More than 3 measurements and ignore values more than 10% from average

(2) Derived variable

(i) CO/CI and SVR are routinely recorded (8 hly or as indicated) on the ICU chart
(ii) Other variables including PVR (I), LVSWI are recorded in the hemodynamics flowsheet
(iii) Oxygen delivery variable are not routinely measured due to limited clinical utility if they are measured oxygen saturation shall be directly measured with co-oximetry
(iv) Table of derived haemodynamic variables

4. Complications

a. Related to CVC cannulation (see CVC section)
b. Related to insertion / use of a PAC
- Tachyarrhythmias
- RBBB
- Cardiac Perforation
- Thromboembolish
- Pulmonary infarction (secondary persistent wedging) ~ 0.1.4%
- Pulmonary artery rupture ~ 0.06-0.2 (mortality 50%)
- Catheter related sepsis
- Endocarditis

- Pulmonary valve insufficiency
- Catheter knotting
- Balloon fragmentation / embolish

F. Pleural drainage

1. Indications
a. Pneumothorax
b. Tension pneumothorax may require urgent needle thoracostomy
c. Haemothorax
d. Large symptomatic pleural effusion

2. Management protocol

a. Needle thoracostomy (tension pneumothorax)
i. 16G cannula placed in mid clavicular line, 2nd intercostals space
ii. Always place an UWSD following this procedure
b. Pleurocentesis (pleural effusion)
i. Local anesthesia and sterile technique

ii. Cannula technique

- 3 way tap attached to 12-14 G IV cannula, syringe and rubber hose (closed system)
- Remove needle from cannula and aspirate pleural effusion until dry

iii. Sedlinger technique

- Pigtail pericardial catheter (preferred) or single lumen CVC kit
- Insert guide wire through needle into pleural space
- Insert catheter into pleural space over wire

Aspirate intermittently with closed system or attaché to an underwater-seal drainage system.

iv. Record volume removed and sends for MC and S, Cytology and Biochemistry

c. Underwater seal drainage

i. Local Anaesthesia in awake patients
ii. Strict aseptic technique at insertion i.e. full gown/glove/mask and cap; *Idophor skin preparation*

Site: Mid axillary line, 3rd – 4th intercostals space

ICU patients need large drains: 28F catheter or larger

Remove trochar from catheter: do not use trochar for insertion of tube
2-3 cm skin incision parallel to the ribs (#10 or #15 scalpel)

Blunt dissection to and through intercostals space with index finger or Howard Kelly forceps until within pleural space

Insert finger into pleural space to enlarge hole and insert tube directly into pleural space or with forceps

Connect to underwater seal apparatus

Insert 2 purse string sutures: 1 to fasten the tube and 1 (untied) to close the incision on removal

Dressing: occlusive dressing (Hypafix)

Check Chest X-ray

xiii. Maintenance

Remove or replace drains inserted in unsterile conditions as soon as possible
Leave drain in situ until radiological resolution, no further bubbling, or drainage (less than 150 ml / 24 hours)
In ventilated patients, drains shall be clamped for 4 hours and removed if none of the above are present

Surgically placed drains (i.e. intraoperative placement) are the responsibility of the surgeon
Streptokinase (STK) Instillation
Only following discussion with the Senior Consultant
This may be done to improve drainage where blockage has occurred due to the presence of blood 250,000UWSD of STK is reconstituted into 50 ml Normal Saline
Instill using aseptic technique
Clear UWSD tubes shall be clamped for 2 hours. Pigtail catheters shall be left open
Patients shall be positioned side-to-side each half hour for 2 hours
The drain shall then be unclamped and drainage volumes notes***

3. Complications (minimized using the blunt technique)

a. Incorrect placement (extrapleural, intrapulmonary, subbiapharagmatic)
b. Pulmonary laceration (haemorrhage, fistula)
c. Pneumothorax
d. Bleeding

- Local incision, intercostals vessels
- Lung
- IMA (with anterior placement)
- Great vessels (rate)

e. Infection: Empyema
f. Mechanical (kinking, luminal obstruction)

G. Endotracheal intubation

1. Policy

a. Endotracheal intubation in ICU patients is a high risk but vital procedure;
i. Usually an emergency procedure, with limited time
ii. Usually indicated for acute respiratory failure, or associated with limited respiratory reserve

iii. Patients may have cardiovascular instability and significant comorbitities
iv. Patients may have cervical spine or oropharyngeal trauma / surgery
v. Patients are at risk of vomiting and aspirating
vi. Positioning is difficult
b. Familiarization with the intubation trolleys, equipment and drugs is essential
c. Intubation shall ideally not be done as a sole operator procedure skilled assistance shall always be sought
d. If you are alone (i.e. after 1730h) **call for help!**
e. The majority of ICU patients mandate rapid sequence induction

2. Indications

a. Institution of mechanical ventilation
b. To maintain an airway
i. Upper airway obstruction

a) Potential for example, early burns
b) Real for example, Epiglottitis, trauma

ii. Patient transportation

c. To protect an airway

i. Patients at risk of aspiration
ii. Altered conscious state
iii. Loss of glottic reflexes

d. Tracheal toilet

3. Techniques

a. Orotracheal intubation is the standard method of intubation in this unit
b. Nasotracheal intubation may be indicated where

i. Patients require short-term ventilation and are intolerant of oral ET tubes

ii. Fibreoptic intubation is indicated
(a) Following head and neck surgery
(b) Inability to open the mouth:
For example, intermaxillary fixation, TMJ trauma, Rheumatoid Arthritis
(c) Upper airway obstruction

iii. Contraindicated in base of skull and LeForte facial fractures

c. Methods

i. Direct visualization under rapid sequence induction
ii. Fibreoptic bronchoscopic awake intubation
iii. Intubating Laryngeal mask – LMA (Fastrac)

4. Endotracheal Tubes

a. Standard tube: Low pressure, high volume PVC oral tube

i. Males 8-9 mm: secure at 21-23 cm to incisors
ii. Females 7-8 mm: secure at 19-21 cm to incisors
iii. Do not cut tubes to less than 26 cm long

b. Double lumen tubes* rarely indicated in ICU

i. Unilateral lung isolation for bronchopulmonary fistula, abscess or haemorrhage
ii. These tubes shall be inserted as a temporary maneuver prior to a definitive procedure
iii. Allow differential lung ventilation

c. Intubated patients from theatre may have the following tubes that are not recommended for prolonged intubation. These tubes shall be changed if intubation anticipated more than 48 hours if safe and feasible

5. Protocol for endotracheal intubation in ICU

a. Personnel intubation is a 4-person procedure, skilled assistance is mandatory:
i. "Top end" intubator who coordinates the intubation
ii. One person to administer drugs
iii. One person to apply Cricoid pressure once induction commences.
- This is recommended as a routine for emergency intubations
- The intubator shall direct the person who is applying Cricoid pressure so that pressure is correctly applied and removed if distortion of the Larynx or difficutly in intubation occurs as a result
- CP is considered safe even in the presence of suspected spinal injury

iv. One person to provide in line cervical spine immobilization (trauma and

spinal patients only). Consider the use of Fastrac LMA for spinal patients to minimize neck movements

b. Secure adequate IV access

c. Equipment: Ensure the following equipment is available and functional

i. Adequate light
ii. Oropharyngeal airways
iii. Working suction with a rigid (Yankauer) sucker
iv. Self inflating hand ventilating assembly and mask
v. 100% Oxygen, i.e. working flow meter at 151/min
vi. 2 working laryngoscopes
vii. Magill forceps
viii. Malleable introducer and gum elastic bougie
ix. 2 endotracheal tubes

a. Normal size and 1 size smaller
b. Check cuff competence

x. Access to difficult intubation equipment

(a) Be aware of failed intubation
(b) Cricothyroidotomy equipment (#15 scalpel / # 6.0 cuffed ETT)

d. Monitoring (on all patients)

i. Pulse oximetry
ii. Blood pressure
iii. Electrocardiography

e. Drugs

i. Induction agent(Thiopentone, Fentanyl, Ketamine, Midazolam)

ii. Suxamethonium (1-2 mg/kg) is the muscle relaxant of choice
(a) Contraindicated in
(1) Burns more than 3 days
(2) Chronic spinal injuries (i.e. spastic plegia)
(3) Chronic neuromuscular disease (for example, Guillain Barre, Motor Neurone Disease)
(4) Hyperkalaemic states (Potassium more than 5.5)
(b) Consider Rocuronium (1-2 mg/kg) if Suxamethonium contraindicated
iii. Atropine (0.6-1.2mg)

f. Procedure: Rapid sequence induction and orotracheal intubation
i. Pre oxygenate with 100% oxygen for 3-4 minutes
ii. Preload with 250-500 ml colloid intravenously
iii. Induction agent and Suxamethonium
iv. Cricoid pressure applied
v. Direct visualization of vocal cords and tracheal intubation
vi. Inflation of cuff until sealed
vii. Confirmation of end-tidal CO2 and chest auscultation with manual ventilation
viii. Cricoid pressure released
ix. Secure tube at correct length
x. Connect patient to ventilator (see default ventilator parameters)
xi. Ensure adequate sedation and muscle relaxant
xii. Consider insertion of a naso/oro-gastric tube. Required by the majority of ICU patients and insertion shall avoid repeating the CXR
xiii. Chest X-ray
xiv. Confirm blood gas analysis and adjust FIO2 accordingly

g. Sedation post intubation
i. None if comat ose or haemodynamically unstable
ii. Morphine / Midazolam, Propofol, Fentanyl, Diazepam as indicated by the clinical scenario

6. Maintenance of endotracheal tubes

a. Tapes
   i. Secure tubes with white tape after insertion
   ii. Ensure that loop of tape is snug around back of neck but not too tight to occlude venous drainage. Shall allow 2 fingers under tape
   iii. Retape with adhesive tape once X-ray check done

b. Cuff checks
   i. Volumetric (sufficient air to obtain a seal + 1 ml) tests are done following, insertion and whenever a leak is detected with a manual hyperinflation once per nursing shift
   ii. Seal is assessed by auscultation over traches during normal ventilation

c. Persistent cuff leaks
   i. Tubes requiring more than 5 ml of air to obtain a seal or if there is a persistent cuff leak shall be examined by direct laryngoscopy as soon as possible even if the tube appears to be taped at the correct distance at the teeth
   ii. Ensure that:
      a. The cuff has not herniated above the cords
      b. Tube has not ballooned inside the oral cavity and "pulled" the cuff above the cords
      c. Patients requiring high airway pressures during ventilation
      d. Aspirate EVAC tubes 2 hourly, or more frequently (hourly) if more than 10 ml supraglottic secretion per hour

7. Endotracheal tube changes protocol

a. Ensure adequate skilled assistance, equipment, drugs and monitoring as for de novo intubation

b. Procedure
   i. Set the FIO2 at 1.0 and change SV modes to SIMV/CMV
   ii. Ensure sufficient anesthesia and muscle relaxation (Fentanyl/ Propofol and neuromuscular blockade)
   iii. Perform laryngoscopy and carefully identify patency of upper airway

after suction, anatomy of Larynx, degree of laryngeal exposure and swelling

iv. Clear view of Larynx and no or minimal laryngeal swelling

a. Application of Cricoid pressure by assistant and careful, graded extubation under direct laryngoscopic vision

b. Maintain laryngoscope and replace tube under direct vision

v. Impaired visualization of Larynx

(a) Use gum elastic or ventilating bougie

(b) Place bougie through tube under direct vision and insert to a length that would be just distal to the end of ETT (approximately 30 cm from end of tube)

(c) Have an assistant control the bougie so that it does not move during movement of the endotracheal tube.

c. Application of Cricoid pressure by assistant and careful, graded extubation

d. Maintain laryngoscopy and ensure bougie is through the cords on extubation

e. Replace tube over bougie and guide through larynx under available vision

vi. Inflate cuff, check end tidal capnography, auscultation, expired tidal volume and then release Cricoid pressure.

vii. Secure tube with tape

8. Extubation protocol

a. Ensure equipment, monitoring and adequate assistance as for intubation

b. Preferentially done during daylight hours and is medical responsibility

c. Extubation criteria

i. Return of equate conscious, state to maintain adequate protective laryngeal reflexes and secretion clearance.

ii. Adequate pulmonary reserve

(a) Respiration rate less than 30 bpm

(b) FVC more than 15 ml/kg

(c) PaO2/FiO2 more than 200

iii. In patients with upper airway surgery or swelling the demonstration of an adequate air leak around the deflated endotracheal tube cuff
iv. Plastic surgical and ENT patients require consultation with the respective specialist. Those patients with intermaxillary fixation and wiring shall have a person from the respective specialist familiar with the placement of the wires and a wire cutter present during extubation

d. All patients shall receive supplemental oxygen post extubation

J. Pericardiocentesis

1. Policy

a. This procedure shall be authorized by the Senior Consultant and performed by the junior medical staff under supervision, or Cardiology
b. Confirmation of pericardial effusion or tamponade shall be made with echocardiography prior to procedure. Liaison with Cardiology is essential

2. Indications

a. Symptomatic pericardial effusion (tamponade)

3. Procedure

a. Strict aseptic technique
b. Local anaesthetic infiltration if awake patient
c. This procedure is greatly facilitated using echocardiography guidance
d. Technique: Sedlinger technique and insertion of a pigtail catheter. Small incision under Xiphisternum

- Insert needle on syringe at 45 degree from the horizontal axis and aim for tip of left Sheller
- Advance slowly and aspirate until confirmation by aspirating blood or serous fluid
- Insert catheter using Sedlinger technique over guidewire
- Confirm placement by aspiration and / or echocardiography
- Check Chest (Pneumothorax)
- Suture and occlusive dressing if leaving for more than 24 hours

4. Complications

- Arrythmias
- Cardiac tamponade
- Myocardial laceration
- Pneumothorax, pneumopericardium
- Liver laceration

K. Intra-aortic balloon counterpulsation

1. Policy

The decision to insert an IABP is made in conjunction with duty Cardiologist and authorized by the Senior Consultant

a. Ischaemic heart disease

- Low cardiac output states following cardiac surgery
- Cardiogenic shock: in association with angiography and revascularization (PTCA, stent or CAVG)
- Acute Mitral incompetence (Papillary Muscle rupture) of VSD following AMI pending operative repair

b. Myocardial disease

- Severe myocardial contusion
- Severe myocarditis
- Cardiomyopathy / Severe Alpha Blocker overdose

2. Contraindications

a. Aortic regurgitation
b. Aortic dissection
c. Severe peripheral vascular disease
d. Tachyarrhythmias (relative)
e. Coagulopathy (relative)

3. Procedure protocol

a. Strict aseptic technique
b. Check IABP function prior to insertion
1) Adequate Helium cylinder to insertion
2) Arterial pressure manifold: referred to mid axillary line and correctly

zeroed

3) Dedicated 5 lead ECG connected to IABP
4) Turn on and leave in standby mode
5) Initial settings

- ECG sense: 1:3 ratio
- Augmentation: minimum (pre-insertion only)
- Inflate and deflate times: zero

c. Insertion procedure

i. Local anesthesia in patients if patient is awake
ii. Scrubbed assistant recommended
iii. Select size (by patient's height)

- Less than 165 cm: 34 balloon
- More than 165 cm: 40 ml balloon

iv. Femoral Artery approach: Sedlinger technique and insertion of a 12f introducer

v. The correct length for insertion shall be checked prior to insertion using the Angle of Louis (level of T4) as the surface landmark. Insert the balloon to the level of T4. The double black marker on the balloon catheter shall be visible – this indicates that the balloon has fully exited the sheath

- Connect to pressure transducer and pump, then press "assist/standby" button to start the pump. Start on minimal augmentation and increase to maximum NB: subsequent augmentation shall not be set below 50%
- Suture in place and cover with an occlusive dressing

- Set timing

a) check balloon inflation against pressure wave set to peak of the dichotic notch
b) Check balloon deflation against ECG: prior to QRS complex and observe decrease in end diastolic pressure
c) Check diastolic augmentation on pressure wave

d) Select augmentation ratio: * standard = 1:1

d. Maintenance

i. Systemic Heparinisation (APTT = 50-80 s)
ii. Check CxR post insertion: tip of IABP below T4 (carina = below the origin of the left subclavian artery
iii. Neurovascular observation of insertion site, lower limbs and left arm hourly
iv. Nurse at less than 30 degree elevation
v. Document pumps timing (ratio) and adequancy of augmentation
vi. Assess haemodynamic response: CI, MAP, SVR, filling pressures, CxR
vii. Nurse clear balloon tubing is exposed, to monitor condensation (due to rapid Helium shutting)

e. Timing during arrhythmias

i. Ectopics: keep on ECG trigger, system shall automatically deflate on ectopic
ii. Tachycardia more than 160/min

a. Reduce augmentation (equal to patient systole)
b. Decrease ratio to 1:2 if reducing augmentation is not adequate

iii. Atrial fibrillation: move deflate slide to extreme right to deflate on R wave
iv. VT or VF: defibrillate or cardiovert as required, the IABP is isolated
v. Cardiac arrest (no output): start ECM

a. Effective output: set on pressure trigger to synchronize balloon inflation with ECM
b. No output: set internal mode for a fixed rate of 40-bpm and 20 ml augmentation

a. Weaning

1) Commence when patient's haemodynamics have improved
2) IABP is generally removed within 72 hours
3) Methods
a. Reducing ratio from 1:1 to 1:2 to 1:3
b. Reduce augmentation. Note: minimum balloon inflation 50%

b. Removal of catheter

1. Notify cardiac / vascular surgeons
2. Cease Heparin 3 hours prior to removal
3. Disconnect IABP tubing: do not aspirate the balloon
4. Use Fem-Stop ® local pressure device immediately on catheter
5. Removal
6. Inflate cuff to 50mm Hg above systolic pressure for 20 minutes

7. Reduce pressure to patient's systolic for 30 minutes
8. Reduce pressure by further 20 mmHg for 20 minutes
9. Remove fem-stop and apply a firm dressing
10. 10-20% cases may require surgical repair to the artery

c. Complications

1. Limb ischemia – thrombotic or embolic
2. Bleeding at the insertion site or systemically
3. Infection
4. Aortic dissection
5. Occlusion of origins of Aortic arch vessels if too high
6. Occlusion of Renal / Splanchnic vessels if to low

7. Thrombocytopaenia
8. Balloon repture: gas embolism

L. Cardiac pacing

1. Policy

- The decision to use transvenous pacing (TVP) is made is conjunction with the duty Cardiologist and authorized by the Senior Consultant
- Become familiar with the theory of insertion, indications, interpretation and complications of TVP

2. Indications

a. Medical pacing the Adrenaline or transthoracic pacing may be adequate to treat many symptomatic bradycardias. Note: this is particularly relevant for retrieveals and has obviated the need for prophylactic pacing in some high-risk patients

b. Any sustained symptomatic bradycardia, which does not respond to medical treatment, or predisposes to a malignant ventricular arrhythmia.

Note : Pacing is indicated by the haemodynamic consequences of the ahythm, not the arrhythmia per se

c. Ventricular tachycardias (especially polyphasic) may respond to overdrive suppression pacing

d. Following cardiac surgery in high-risk patients (epicardial leads)

- Valve replacement / Repair: Especially Mitral
- VSD repair / Papillary Muscle rupture
- Acute Myocardial Infarction

3. Types

a. Bipolar pacing lead (VVI): insert under image intensification
b. Balloon flotation leads: may be inserted under ECG or pressure guidance
c. Paceport PA catheters: these have little utility
d. Epicardial leads
e. Placed during cardiac surgery in high risk patients
f. Usually unipolar ventricular, but may be bipolar, atrial or ventricular: check the operative note and liaise with the surgeon

4. Procedure protocol (VVI Lead)

a. Strict aseptic technique
b. Image intensification
c. Local anesthesia where appropriate
d. Insertion protocol

1. 6F peel away sheath or PAC introducer
2. Right Internal Jugular Vein is the preferred route, then left subclavian

NB: If permanent pacing is likely then avoid Subclavian placement

3. Under I-I control, feed the wire through the RA until the tip just stops on the right ventricular wall
4. Connect to the control box (switched off)
5. Set output and sense to their minimum value, and rate 20bpm faster than the patient's own rate (or 70bmp, whichever is greater)
6. Turn the generator on and gradually increase the output while watching the ECG for capture
7. If there is no capture or a high output is required

a. Place on demand mode
b. Turn output right down, advance or reposition the wire slightly
c. Try to capture again. An ideal capture setting is ~ 2 MA
d. Ensure wires not exposed and tape both sides

ii. Suture the wire and apply an occlusive dressing
iii. Arrange a post-insertion CxR

e. Daily check

i. Battery strength
ii. Capture: set the output 2 x higher than threshold for safety

5. Floatation Catheter Insertion

a. These may be inserted either "blind", under ECG guidance (standard recommendation), or via pressure guidance for catheters having an infusion lumen (cf.PA catheter insertion)

b. Aseptic technique and local anesthesia where appropriate

c. Insertion protocol

1. 6F peel away sheath, do not use a PAC introducer as these shall leak
2. Attach V5 lead of an ECG to the distal electrode of catheter and monitor
3. Note P and then QRS waveform changes as the catheter advances to the RV
5. Advance catheter another 2cm, deflate the balloon and advance 1cm
6. Connect to the pulse generator (switched off)
7. Set output and sense to their minimum value, and rate 20bpm faster than the patient's own rate
8. Turn the generator on and gradually increase output while watching the ECG for capture
9. If there is no capture or a high output is required – sec(4.d.7) above
10. Suture the wire and apply an occlusive dressing
11. Arrange a post-insertion CxR

7. DRUGS AND INFUSIONS, HEPARIN PROTOCOL

1. Policy

1. Patients admitted to ICU shall have all old and current medications reviewed. Only medications that are applicable to the current admission shall be transcribed to the Drug Chart
2. All drugs, infusions and fluids are reviewed and written up daily
3. All subsequent changes or additions to drug and fluid orders shall be written and signed for on the Drug Chart. Nursing staff shall be notified of these changes or additions as soon as charted. Verbal orders alone are neither sufficient nor legal
4. Vasoactive or hypertonic infusions (for example, TPN) shall be administrated through dedicated lumens of a central venous catheter
5. The concentration of infusions shall not be changed (i.e. 'double strength') from the unit infusion protocols outlines below. Standardization of infusion concentrations is an essential quality control exercise to prevent confusion and potentially dangerous drugs mishaps
6. Charting of drugs and infusions is only to be done by Intensive Care Medical Staff
7. Patients discharged from ICU shall have appropriate drugs, infusions and fluids written up on standard hospital forms
8. Notify the Anesthesia Department about patients discharged under their care (i.e. epidurals, PCA)

Principles of drug prescription in Intensive Care

1. Ideally, drugs shall only be prescribed where proven benefit has been demonstrated
2. Drugs shall be prescribed according to protocols and guidelines
3. Ensure that the drug doses are correct: Check the Formulary if unsure

4. The risk and benefit of starting any drug shall be carefully considered. Critically ill patients have altered pharmacokinetics and pharmacodynamics with the potential for toxicity and drug interactions
5. Where possible:
   - Use drugs that can be titrated or prescribed to an easily measured endpoint
   - Use drugs that can be measured to monitor therapeutic drug levels

   - Avoid drugs with narrow therapeutic indices (For example, Digoxin, Theophylline), particularly in patients with associated Hepatic or Renal dysfunction
   - Cease a drug if there is no apparent benefit
   - If two drugs are of equal efficacy, choose the cheaper drug (For example, Pancuronium vs. Vecuronium) as the cost of drugs in ICU is significant.

Cardiovascular Drugs

1. Inotropes

Inotropes (specifically catecholamines) are the most frequently used cardiotropic drugs in intensive care. Despite this, there are varied prescription practices and preferences for these drugs, which are usually based on the purported effects of the different agents.

a. General principles

- Maintenance of blood pressure in critically ill patients forms the basis haemodynamic resuscitation and organ perfusion
- Hypovolaemia is the most common cause of hypotension and low cardiac output in critically ill patients and shall be assiduously monitored and corrected
- The main indication for inotropes is to increase myocardial contractility for a given preload and after load. Clinically this

equates with low mean arterial pressure (MAP) or cardiac output states

- MAP and CO shall be interpreted in the context of pre-morbid cardiac function and the expected response to an acute insult
- The use of inotropes in other than trivial doses requires regular haemodynamic monitoring (arterial line and CVC) and where indicated a PA catheter to assess flow (CO) as well as pressure
- Inotropes primarily increase cardiac output and mean arterial pressure. These agents have variable effects on heart rate and peripheral muscular resistance (systemic and pulmonary)
- No single inotrope (or mixture of inotropes) has been shown to be superior to another
- There is marked inter-individual variation in the response to inotropes. This is partly due to qualitative changes it. Adrenergic receptor kinetics in acute illness. Similarly, prolonged exposure to Catecholamine infusions may produce adrenergic receptor down-regulation

- The clinical implications are

(a) Catecholamines (both endogenous and exogenous) act in similar fashion with Alpha Adrenergic effects predominating at low doses and Beta

(b) It is impossible to predict the dose range for an individual patient and prescription of inotropic infusions on a body weight basis (µg/kg/min) is of little clinical utility

(c) Infusions shall be started at a low rate (3-5 µg/min) and increased until a satisfactory clinical response occurs

(1) Improvement in MAP, peripheral perfusion and CO
(2) No significant increase in HR or development of arrhythmias
(3) Maintenance or increase in urine output
(4) Reversal of acidosis

(d) High concentration of inotropes may be required to achieve the desired effect (more than 50 µg/min)

ADRENALINE

INDICATIONS:

1. Cardiopulmonary resuscitation
2. Severe sepsis syndrome, septic shock
3. Cardiogenic shock
4. Anaphylaxis
5. Maintenance of cerebral perfusion pressure
6. Medical pacing

NORADRENALINE

MONITORING

- Continuous ECG, BP
- Fluid Balance
- Assess extremities for Change in Colour or temperature
- Assess IV site for sign's of extravasation, area will appear cold, hard and pale

ADVERSE EFFECTS:

Severe peripheral and visceral varo constriction associated with hypovolemia decreased renal perfusion and decrease urine output tissue hypoxia and metabolic acidosis

- Plasma volume depletion, associated with prolonged use
- Decrease cardiac output
- Hypertension, reflex bradycardia
- Arrhythmias
- Anxiety or restlessness, dizziness, headache, trembling

EXTRA VASATION: Result is sloughing and necrosis

Treatment: stop infusion

Restart at new site; notify physician infiltrate the area with Phentolamine 5 to 10 mg diluted in 10 ml NS
Paediatrics: 2.5 mg or Use nitroglycerin 2% ointment

DOSE:

Adult: 8-12 MCG/MIN or 0.01-0.5MCG/kg/MIN

Maintenance: 2-4MCG/MIN or 0.03-1.5 MCG/kg/MIN

Pediatric: 0.05-0.1 MCG/kg/MIN

Maintenance: 2MCG/kg/MI

DOPAMINE

MONITORING:
1. Continuous ECG and BP
2. Cardiac output monitoring
3. Assess IV Site for Sign and symptoms of extravasations; area will appear cold, hard and pale
4. Assess Extremities for change in colour or temperature

ADVERSE EFFECTS :

- Increase heart rate ectopic beats, palpitations, ventricular arrhythmias
- Angina nausea vomiting (rare) vasoconstriction (Dose>10MCG/kg/MIN, can cause hypertension headache dyspnea vascular stasis and gangrene in extremities)

EXTRAVASATION:

- May results in sloughing and tissue necrosis
- Use central line or large veins e.g. cephalic or basilar, to decrease risk

Treatment:

- Stop infusion
- Restart at new IV site and notify physician infiltrate area of extravasations with phentolamine. 5-10mg diluted in 10ml NS (adults) 0.1-0.2mg/kg up to 10 mg diluted in 10 ml NS (pediatrics) use a fineneedle or use Nitroglycerine 2% ointment local application

DOBUTAMINE:

MONITORING:

- ECG and BP
- Cardiac output
- Fluid balance
- Serum Potassium

ADVERSE EFFECT:

- Ventricular ectopic beats, increased hearts rate, hypotension
- Non specific chest pain, shortness of breath, palpitations
- Decreased serum potassium concentration, nausea, vomiting
- Headache
- Hypersensitivity reactions including rash, fever, bronchospasm (rare)

DOSE:

- **ADULTS:** Usual initial dose is 2-3 MCG/kg/min Increase in 2-3MCG/kg/min increments at 10-30 min intervals according to response. Usual range is 7.5-15MCG/kg/min

- **PEDIATRIC:** 2-20 MCG/kg/min Adjust according to response

VASOPRESSIN

ADD 40 UNITS IN 40 ml NS or 5% DEXTROSE

- 0.02 units/min = 1.2 ml/hr
- 0.03 units/min = 1.8 ml/hr
- 0.04 units/min = 2.4 ml/hr
- 0.05 units/min = 3 ml/hr
- 0.1 units/min = 6ml/hr

3. Antihypertensive agents

a. General principles

i. The most common cause of hypertension in Intensive Care patients is sympathetic drive due to pain agitation or delirium. This shall be treated with adequate sedation and analgesia

ii. Patients is the recovery phase of acute renal failures are other hypertensive and as such do not need treatment

iii. Similarly, neurogenic hypertension is frequent following head injury or intracerebral haemorrhage and is generally self-limiting and dose not require treatment. The use of vasodilators in this setting is relatively contraindicated

iv. Antihypertensive shall be titrated against the patient's premorbid blood pressure

b. Indications

i. Acute

(a) Acute perioperative control of hypertension after cardiac, carotid, or cerebrovascular surgery, or for patients with critical myocardial ischemia

(b) Hypertensive crisis (malignant hypertension)

(c) Pre-eclampsia / Eclampsia

ii. Other indications for vasodilators

(a) Reduction of afterload in cardiac ischemia and failure

(b) Adjunct to passive warming in hypothermia

iii. Chronic

(a) Reflex tachycardia

(b) Hypotension (especially in Hypovolaemic patients)

(c) Tachyphylaxis

(d) Pulmonary vasodilatation causing shunt and hypoxia

(e) Cyanide toxicity (SNP)

NITROGLYCERIN

Add 5 mg nitroglycerin in 50 ml 5%Dextrose or NS

OR

Add 50 mg nitroglycerin 500 ml 5%Dextrose or NS

Dose

The usual starting dose is 5µg/min every 3-5 minutes until desired response is obtained. If no effect is obtained with 20mcg/min, the infusion maybe increased by 10mcg/min increments. The usual maximum dose is 200mcg/min

When discontinuing a drip, decrease dosage by 5mcg.min increment and carefully monitor patient response

Monitoring: Continuous BP, HR, RR and ECG

Nitroglycerin 5 mg in 50 ml (100mcg/ml)

OR

Nitroglycerin 50 mg in 500 ml (100mcg/ml)

DOSE	INFUSION RATE
5 mcg/min	3ml/hr
10 mcg/min	6ml/hr
15 mcg/min	9ml/hr
20 mcg/min	12ml/hr
30 mcg/min	18ml/hr
40 mcg/min	24ml/hr
50 mcg/min	30ml/hr
60 mcg/min	36ml/hr
70 mcg/min	42ml/hr
80 mcg/min	48ml/hr
90 mcg/min	54ml/hr
100 mcg/min	60ml/hr
110 mcg/min	66ml/hr
120 mcg/min	72ml/hr
130 mcg/min	78ml/hr
140 mcg/min	84ml/hr
150 mcg/min	90ml/hr
160 mcg/min	96ml/hr
170 mcg/min	102ml/hr
180 mcg/min	108ml/hr
190 mcg/min	114ml/hr
200 mcg/min	120ml/hr

CONTRAINDICATIONS

- Hypotension or uncorrected hypovolemia
- Increased intracranial pressure (e.g. head trauma or cerebral hemorrhages)
- Constrictive percarditis or pericardial tamponade

- Use of sildenafil within previous 24 hours may cause irreversible hypotension

ADVERSE REACTIONS

- Hypotension
- Headache, flushing, Dizziness
- Nausea, Vomiting
- Bradycardia
- Methemoglobinemia with very large doses (blue skin and mucous membranes, vomiting, shock, coma)

SODIUM NITROPRUSSIDE:

(Maximum 200 mg in 500 ML 5% Dextrose or NS)
Dose: 0.5-8mcg/kg/min

Start with 0.5 mcg / kg/ min, then the dose may be titrated up slowly by 0.5MCG/KG/MIN increments every 5 minutes until the desired pressure is achieved

DILUTION: Add 2-3 ml 5% dextrose or OR sterile water in 50 mg nitropresside
Then added this in 500 ml 5% dextrose of NS
Freshly prepared solutions have faint brownish tint; nitroprusside decomposes on exposure to light. It will change from a light brown to a dark brown, orange or blue

1. Protect from light
2. Discard the solution if it turns blue green or dark brown

MONITORING:

1. Continuous BP, HR, urine out put

2. Daily serum thiocyanate conc. In those receiving prolonged infusion (>2 days) of > 3mcg/kg/min or renal impairments

ADVERSE REACTIONS:

1. Profound hypotension
2. Thyocyanate toxicity, which may cause a neurotoxic syndrome and symptoms of hypothyroidism
3. Cyanide toxicity (metabolic acidosis is an early indicator)
4. Nausea, Vomiting, diaphoresis, nasal stuffiness, muscular twitching dizziness, weakness

LABETALOL:

Dose: - An initial dose of 10 mg slow IV
Additional 10 mg or 40 mg can be given at 10min intervals until the desired supine Bp is achieved or total of 300mg/24 hr has been given

FOR IV INFUSION

Infuse up to 2 mg / min or 120 ml / hr to obtain Desired BP Control
Add 20 ml labetalol (100mg) in 30 ml normal saline = 100mg/50 ml

DOSE	INFUSION RATE
0.5 MG/MIN	15 ML/HR
1MG/MIN	30ML/HR
1.5MG/MIN	45ML/HR
2MG/MIN	60ML/HR

MONITORING:

CONTINUOUS BP, HR, ECG

Due to potential for postural hypotension and fainting during the initial 3 hr post dose the patient's ability to tolerate the up right position should be established prior to first ambulation

ADVERSE REACTIONS: Hypotension, breathing difficulty, CHF irregular heart beat Bradycardia, dizziness

Agent	Standard infusion/dose	Uses
Phentolamine	10 mg/ 10 ml 5 % Dextrose titrate	1. Pure alpha blockage , Short acting anti hypertensive
Hydrallazine	10-20 mg as bolus 20-40 mg 6-8 hourly	1. Short to medium term parenteral antihypertensive 2. Often use with alpha blockers for reflex tachycardia 3. Useful in renovascular hypertension
Amplodipine	5-10 mg oral bd	1. Long acting oral calcium antagonist 2. Caution in renal failure
Captopril	Start low dose 3-6.25 mg upto 50 mg orally 8 hourly Syrup: 5 mg/ml or tablets Acute hypertension : 6.25-25 mg sublingually prn	1. Treatment of hypertension 2. Left ventricular dysfunction, especially post -MI 3. Left ventricular failure 4. Diabetic Nephropathy 5. Caution in Reno vascular disease and Renal Failure
Enalapril	Start 2.5 mg daily upto 20 mg bd orally	1. Treatment of hypertension 2. Left ventricular dysfunction, especially post -MI 3. Left ventricular failure 4. Diabetic Nephropathy 5. Caution in Reno vascular disease and Renal Failure
Phenoxy-benzamine	Oral : 10 mg/day untilpostural hypotension IV :1 mg/kg/day (dilute to 200-500 ml): 1/3 dose over 1/24,2/3 dose over	1. Long acting alpha blocker 2. Preoperative preparation of phaneochromocytoma 3.Idiosyncratic Hypotension may occur

	1/24	
Prazosin	Start with 0.5 mg, and increase upto 5 mg bd orally	1. Alpha blocker 2.Potent Antihypertensive agent 3. Beware first dose effect,especially under filled
Atenolol	1-2 mg IV bolus (upto 10 mg) Oral: 25-100 mg bd	1.Used in high sympatehtic drive states: neurogenic hypertension 2.Allgrades of hypertension , including reno vascular. 3. Cardiac ischemia 4. Control of reflex Tachycardia with vasodilators 5. Thyroid crisis 6. Caution in poor LV Function, Asthma 7.Renal excretion; Virtually no Hepatic metabolism
Metoprolol	Oral: 25-100mg bd	1. As for atenolol 2.Mainly eliminated by hepatic metabolism
Esmolol	Loading Dose 0.5 mg/kg Infuse 100 mg/10 ml and titrate	1.Ultra short acting Alpha-blocker 2.Useful as trial for patients with poor LV function 3. Adjunct to vasodilators post cardiac surgery
Clonidine	Start 25ug@ 150 ug IV Oral :75 ug bd Upto 150-300 ug tds	1.acute Centrally mediated hypertension 2. Useful post cardiac surgery 3 Withdrawal states 4. Care with Hepatic or Renal Dysfunction 5 May cause rebound Hypertension with chronic use
Methyldopa	250-2g/day oral bd 125 mg-250mg IV bolus	1.Third line drug for chronic hypertension, especially if tolerant of ACEI 2.Pregnancy induced Hypertension 3. may require diuretic/ACEI 4 IV useful in centrally mediated hypertension

4 Antiarrhythmics

General Principles

I. Prior to administration of antiarrhythmic agents, optimize correction of the following
   a. Hypovolaemia
   b. Metabolic abnormalities (Decreased Calcium, Magnesium and Biophosphate leading to alkalosis)
   c. Myocardial ischemia or cardiac failure (especially post cardiac surgery)
   d. Sepsis
   e. Pain and agitation

ii. All antiarrhythmic drugs shall be regarded as pro-arrhythmic

iii. Virtually all depress myocardial contractility

iv. Antiarrhythmics drugs are indicated when the arrhythmia causes haemodynamic compromise (hypotension or prolonged Tachycardia more than 120-130 / min) or in susceptible patients with Myocardial Ischemia

b. Indications

i. Termination of an acute arrhythmia

ii. Prophylaxis for recurrence

AMIODARONE

(150 mg/3ml ampoule)

DOSE:

Loading dose:

Prepare solution with 150 mg Amiodarone in 100ml 5% dextrose in non PVC bag

Infuse 150 mg over 10 minutes

Maintainence 1mg/min for 6 hours

Prepare solution with 900mg Amiodarone in 500 ml 5% dextrose in glass bottle or non PVC bag
Infusion at a rate of 33.3ml/hour for 6 hours
(Infusion of 360 mg over the next 6 hours, 1mg/min)

Maintainence of 0.5 mg/min over next 18 hours
Infuse at a rate of 16.7ml/hour over next 18 hours
(Infusion of 540 mg over the remaining 18 hours, 0.5 mg/min)

Loading dose:- 150mg in 100 ml 5% dextrose over 10 min then 33.3 ml/hour for 6 hours then 16.7 ml/hour for 18 hours

If break through episodes of ventricular fibrillation or tachycardia occur, an additional infusion of 150 mg over 10 min may be administered

- When switching from IV to PO
- <1 week IV :- 800-1600mg/day
- 1-3 weeks IV:- 600-800mg/day
- >3 weeks IV:- 400mg/day

AMIODARONE

Monitoring:

- Continuous ECG
- BP, HR
- Monitoring peripheral site for pain, redness, swelling every fourth hourly

- Serum electrolytes and acid base balance
- Liver Enzymes
- Pulmonary Function Test
- Serum Creatinine
- Thyroid Function Test

Adverse Reactions:

Cardiovascular:- Sinus Brady Cardia, Hypotension, Heart Block, Proarrhythmic effects.
Pulmonary:- Pulmonary fibrosis, interstitial pneumonitis (fever, shortness of breath, cough)
Thyroid: Hypo or Hyper thyroidism
Gastro Intestinal: Nausea, Vomiting, Anorexia, Abdominal Pain, Constipation
Hepatic: Abnormal liver function tests

XYLOCARD (LINGOCAINE)

Dose:

Initial bolus:

- 1mg/kg/at a rate of 25-50mg/min
- May repeat ½ of original dose in 5-10 minutes if needed
- Not to exceed 300 mg in 1 hr

BOLUS	40 KG	45 KG	50 KG	55 KG	60 KG	65 KG	70 KG	75 KG	80 KG	85 KG	90 KG
DOSE IN ML	2 ML	2.25 ML	2.5 ML	2.75 ML	3 ML	3.25 ML	3.5 ML	3.75 ML	4 ML	4.25 ML	4.5 ML

Give bolus dose over 2-3 min

- For continuous infusion

- Take 2% 50 ml of undiluted solution of xylocard in 5% dextrose or normal saline
- Infusion of 1-4mg/min after IV bolus

Xylocard 2% 50 ml of undiluted xylocard (conc: 20mg/ml)

TABLE – 9

Dose	Infusion rate
1mg/min	3 ml/hr
2mg/min	6 ml/hr
3mg/min	9 ml/hr
4mg/min	12 ml/hr

Monitoring:

- Continuous ECG
- BP, HR
- CNS toxicity – ringing in ear circumoral numbness, metallic taste, nausea dizziness, sedation
- Serum electrolytes

Adverse reactions:

1. Cardio vascular: hypotension, amhythmias, heart block, cardiovascular collapse, Bradycardia
2. CNS – confusion, agitation, nausea, vomiting, convulsions, coma and death
3. Somnolence, paresthesia

Thrombolytic Thearpy

NB: All patients with acute myocardial infarction shall be considered as potential candidates for primary angioplasty. The Cardiology cover shall therefore be notified as early as possible of all patients with acute MI. The

duty Cardiologist shall decide between primary angioplasty, thrombolysis, and expectant management

a. Indications
i. Acute Myocardial Infarction
a. Thrombolytic therapy is now standard in the management of acute Myocardial infarction, however, the patient shall be advised of the potential risks and benefits.
b. Administered in consultations with the duty Cardiologist
c. Patient criteria
1. No specific age limit – discuss with Cardiology
2. Ischaemic chest pain for more than 30 min and less than 12 hrs
i. Benefit is inversely proportional to delay in thrombolysis, thus therapy shall be considered a "medical emergency"
ii. Late therapy may be inappropriate for "small "infarcts
3. ECG evidence of acute infarction
i. Raised ST segment 2mm in two (or more) chest leads, or
ii. Raised ST segment 1mm in two adjacent limb leads, or
iii. New LBBB
iv. Posterior infarction (Raised R in V1 and depressed ST in V2)

Note: No benefit has been demonstrated for patients with ST depression, T-wave inversion, or a normal ECG

4. Haemodynamically significant pulmonary embolism
a. An unequivocal diagnosis (usually spiral CT or Angiogram) is necessary prior to thrombolysis
b. Tenecteplase is preferred in life threatening pulmonary embolish

b. Contraindications
i. Absolute
a. Lack of verbal informed consent
b. Active internal bleeding

c. Recent head trauma, major trauma or surgery within 3 weeks
d. Refractory hypertension (more than 200/100 mmHg)

ii. Relative
a. Known bleeding diathesis
b. Current use of anticoagulants (WARFARIN)
c. Active peptic ulceration or other GI bleeding within 6 months
d. Prolonged CPR (more than 10 mins) and / or traumatic resuscitation
e. Pregnancy
f. Diabetic Proliferative Retinopathy
g. Non-compressible vascular puncture / injury

iii. Each of these relative contraindications shall be considered in view of the potential clinical benefit and risk to each patient

c. Complications
i. Bleeding (incidence of Cerebral hemorrhage is ~ 0.5%)
ii. Anaphylaxis/anaphylactoid reactions: hypotension, rash bronchospasm
iii. Reperfusion arrhythmias

d. Routine follow-up
i. ECG at and 4 hours post STK/TNK
ii. Cardiac enzymes 6, 12 and 24 hours post infusion
iii. If ST-elevation persists 1 hr post – TNK, contact cardiology regarding "rescue angioplasty"

Agent	Standard infusion/dose	Uses

Streptokinase	1.5 million units/ 50 mls normal saline , over 60 minutes via syringe pump	1. 150 mg Aspirin prior to therapy, then daily 2. No 1 Heparin 3. Heparin 12,500 u S/C 4 hours post commencement of STK then bd for 14 doses 4. STK can be re-used within 3 days for re-infraction.
Bleeding Protocol	Monitor APTT PR Fibrinogen level Euglobulin clot lysis time	1. Local Pressure 2. Reverse Heparin with Protamine (Check APTT) 3 Cryoprecipitate 10 units along with FFP 2 units 4. Defribination or Intracranial bleed EACA : 5 g over 1 hr then 1g/hr

5 Antiplatelet Agent

Agent	Standard infusion/dose	Uses
Reopro (abciximab)	Bolus :0.25 mg/kg IV over 1 min, 10 mins before PTCA Infusion 0.125 ug/kg/min IV for 12 hrs.(max rate =10 ug/min)	Only to be ordered by cardiology Binds to platelet Glycoprotein Hb/III a receptor, Inhibiting platelet aggregation and thrombus formation Primarily used with PTCA Used with Aspirin and Heparin (although no set protocol for Heparin) increased risk of major bleeding
Tirofiban	Bolus : 0.4 ug/kg/min for 30 mins maintenance : 0.1 ug/kg/min for atleast 48 hrs NB: reduce doses by 50 % with severe renal insufficiency (for eg, Creatinine clearance less than 30 ml/min)	Only to be ordered by cardiology. Block platelet Glycoprotein Iib/III a receptor. Short Half-life (1.4-1.8 hrs) Uses :unstable angina, non-Q wave MI Use with Heparin and Aspirin continue through Angiography, and for 12-24 hrs post -PTCA Check Platelet count 6 hrs postbolus, then atleast daily SES: Bleeding (major 1.4%) ,thrombocytopaenia,fever
Clopidogrel	75 mg orally daily 300 mg oral loading dose pre-PTCA (then 75 mg daily)	Irreversibly modifies platelet ADP Receptor, inhibiting aggregation Uses: Prevention of vascular ischaemic events for example MI, CVA, PTCA
Integrilin	90-250 ugm/kg	Non-Qwave MI and as adjunct to PTCA

	2 ugm/kg/minute	

D. Respiratory drugs

1. Nebulised bronchodilators

a. General Principles

i. These agents are the mainstay of treatment for bronchospasm in Intensive Care (including acute severe asthma) They are not routinely used in all ventilated patients.
ii. Once commenced, they shall be reviewed daily regarding efficacy. This is assessed by improvements in audible wheeze, lung compliance, respiratory rate and blood gases

b. Indications

Pre-existing asthma/chronic airflow obstruction **(CAO)**

- Acute severe asthma
- Acute bronchospasm secondary to infection, aspiration or during mechanical ventilation
- Acute exacerbation of CAO

2. Parenteral Therapy

a. Indications

i. Ajunctive therapy for acute severe asthma in patients not responding to Nebulised agents
ii. Selected patients who are difficult to wean from ventilation (usually due to CAO)
iii. Maintenance in patients with chronic airflow obstruction

b. Complications

i. Hypokalaemia, metabolic alkalosis
ii. Arrhythmias (Theophylline)
iii. Intercurrent information (steroids)
iv. Polyneuropathy (steroids)

Agent	Standard Infusion/Dose	Uses
Salbutamol (nebulised)	Nebulised in Normal saline (1ml:1ml) continously , 2 or 4 hourly	1. First line bronchodilator 2. Used in severe hyperkalemia
Ipratroprium Bromide	Nebulised in addition to salbutamol (1ml:1ml)	1. Chronic airflow obstruction (CAO) 2. Bronchorrhoea
Budesonide (nebulised steroid)	Nebulised 1 mg bd	1. Steroid dependent (CAO) 2 Acute exacerbation of CAO
Salbutamol(IV)	6mg/100 ml 5 %Dextrose (ml/hr=g/min)	1.Acute severe asthma 2 Longer duration of action
Hydrocortisone	100mg IV 4-8 hourly	1. All patients with acute severe asthma;wean off over 48-72 hours once broken 2. Acute exacerbation of CAO

AMINOPHYLLINE:
ADULT: Loading Dose
Patient not currently receiving Theophylline products =6 mg/kg
Patients currently receiving theophylline products = no loading dose

Maintenance dose:

- Otherwise healthy non-smoking adult 0.7 mg/kg/hour
- Patients over 60 or with cor pulmonale 0.25 mg/kg/hour

- Patients with CHF or liver disease 0.25 mg/kg/hour
- Young smoking adults 0.9mg/kg/hour

For maintenance add 500 mg (2 amps) aminophylline in 480 ml 5% dextrose or NS

Patient weight in KG	**Loading Dose**	**Maintenance Dose**		
		0.25 mg/kg/hr	0.7mg/kg/hr	0.9 mg/kg/hr
40	240mg	10	28	36
45	270 mg	11.25	31.35	40.5
50	300 mg	12.5	35	45
55	330 mg	13.75	38.5	49.5
60	360 mg	15	42	54
65	390 mg	16.25	45.5	58.5
70	420 mg	17.5	49	63
75	450 mg	18.75	52.5	67.5
80	480 mg	20	56	72
85	510 mg	21.25	59.5	76.5
90	540 mg	22.5	63	81
		ML/HOUR		

Or
For Maintenance add 250 mg Aminophylline in 50 ml NS[5mg/ml]

Pt.Weight in kgs	**0.25 mg/kg/hr**	**0.7mg/kg/hr**	**0.9 mg/kg/hr**
40	2 ml/hr	5.6 ml/hr	7.2 ml/ hr
45	2.25ml/hr	6.3 ml/hr	8.1 ml/hr

50	2.5 ml/hr	7 ml/hr	9 ml/ hr
55	2.75ml/hr	7.7 ml/hr	9.9 ml/hr
60	3 ml/hr	8.4 ml/hr	10.8 ml/hr
65	3.25 ml/hr	9.1 ml/hr	11.7 ml/hr
70	3.5 ml/hr	9.8 ml/hr	12.6 ml/hr
75	3.75 ml/hr	10.5 ml/hr	13.5 ml/hr
80	4 ml/hr	11.2 ml/hr	14.4 ml/hr
85	4.25 ml/hr	11.9 ml/hr	15.3 ml/hr
90	4.5 ml/hr	12.6 ml/hr	16.2 ml/hr

Adverse Reactions:

- Serum levels > 20mcg/ml: nausea, vomiting, diarrhea, headache, insomnia, irritability
- Serum levels > 35 mcg/ml: hyperglycemia, hypotension, cardiac arrhythmias tachycardia
- CNS – irritability, restlessness, headache, insomnia, reflex hyperexcitability, muscle twitching, convulsions
- G I – Nausea, vomiting, epigastria pain, hematemesis, diarrhea, gastro-esophageal reflux

- C V S – Palpitation, tachycardia, extrasystole, hypotension, circulatory failure, ventricular arrhythmias
- Tacypnea, proteinuria, fever, hyperglycemia, rash

Monitoring:

- BP, heart rate, respiratory rate

- Serum levels

ANALGESICS AND SEDATIVES

Agent	Standard Infusion/Dose	Uses
Midazolam	30 mg per 50 ml 5% Dextrose Rate :1-30 ml/hr	1. Standard Sedation regimen 2. Review rate/ Sedation at least daily 3.Effects prolonged in Renal Failure
Morphine	1-5 mg IV, sc prn	Maybe Useful in pulmonary edema
Fentanyl	100-200 Micg IV bolus infusion:50-200 micg/hr(Neat Solution)	1. Haemodynamically Stability 2 . Potent Medium acting narcotic 3. Useful for ICU procedure
Diazepam	IV : 2-10 mg/prn Orally :Usually 5-10 mg bd-qid*	1.First Line anticonvulsant(IV) 2. First Line anxiolytic(in delirium)
Propofol	10 mg/ml(neat solution), start at 3ml/hr and titrate against effect	1 .Short term sedation of intubated and ventilated patients, where extubation is expected within 24-48 hours 2. Do not use in patients where prolonged ventilation is anticipated, except where repeated neurological assessment is required as in CHI or in the presence of Hepatic or Renal failure 3. Anaesthesia for minor procedures where prompt return of consciousness is required(tracheostomy,CVC) 4 .Potent myocardial Depressant 5 No analgesic effect
Haloperidol	2.5-5mg IV prn	1 First Line major tranquilizer,use for delirium, agitation as in opioid/Benzodiazepine withdrawal 2 .Blocker : may cause hypotension
Chlorpromazine	5-10 mg IV prn	1 As per Haloperidol; 2nd line tranquiliser 2 More sedating, unpredictable and longer acting agent
Lorazepam		Used in status Epilepticus and as an anxiolytic in pre-anesthetic medicine

2 Muscle Relaxants

a. General Principles

I. The use of muscle relaxants for the critically ill patients is different to patients undergoing elective anesthesia.

II. These agents have a limited role in Intensive Care and shall not be used unless the patient is adequately sedated

III. Non-deplorizing agents (except Rocuronium) shall not be for emergency (rapid sequence induction) Endotracheal intubation

b. Indications

i. Depolarizing: Suxamethonium
   (a) First line relaxant for emergency Endotracheal intubation

ii. Non-depolarising: Pancuronium, Vecuronium, Atracurium
   (a) Acute control of ventilation post inbutation
   (b) Patient transport / retrieval on Oxylog ventilator
   (c) Selected patients with poor lung complications
   (d) Acute procedures: tracheostomy, bronchoscopy

c. Complications

i. Hyperkalemia, Bradycardia (Suxamethonium)
ii. Polyneuropathy (especially with concomitant use of steroids)
iii. Sympathetic overdrive, particularly in under-sedated patients
iv. Adverse outcome in head injury when used as a measure to control ICP

TABLE 16

Relaxant	Dose	Comment
Suxamethonium	100-200 mg or 1-2 mg/kg	1. 1st line agent in Rapid Sequence Induction (RSI) 2. Consider pre-treatment with atropine (0.6-1.2 mg) if potential Bradycardia 3. Contraindicated in burns (More than 3

		days), chronic spinal and neuromuscular disease, hyperkalemic states (Potassium more than 5.5)
Rocuronium	0.6 mg/kg 1.0 mg/kg for RSI	1. First line non-depolarizing agent in ICU 2. Rapid onset (60 sec) 2nd line agent in RSI-used as alternative to Suxamethonium 3. Duration of action 30-40 minutes
Pancuronium	2-4 mg IV prn	1. 2nd line non-depolarizing agent in ICU 2. Long duration of action 3. Tachycardia rarely a problem in ICU patients

F. Anticoagulants

1. General principles

a. Anticoagulants are potentially dangerous drugs
b. All patients on systemic anticoagulation shall have an APTT, INR and CBP performed daily

2. Indications
a. Acute systemic anticoagulation
i. As a general rule, Heparin infusion titrated to a therapeutic APTT is used in critically ill patients. This allows monitoring and the provision for reversal if indicated (for example, procedures, bleeding complications)
ii. High dose Enoxaparin (for example, 1mg/kg bd s/c) is as effective, but potentially more difficult to use in critically ill patients, due to inability to easily monitor activity and reverse effect
a. Proven venous or arterial thromboembolish
b. Myocardial infarction: as sole therapy or with following PA Prosthetic heart valves
1. Prior to commitment of oral anticoagulants

2. During an acute illness, where oral anticoagulation is relatively contraindicated
c. AF in patients complicated by emboli less than 70 years
d. AF for more than 48 hours, in which cardio version is being considered
e. Extracorporeal circuits. For example, CVVHDF
f. IABP

b. Partial anticoagulation (low dose IV Heparin (500 u/hr), IV Prostacyclin)

c. Patency of CVVHDF circuits (always consider Heparin free circuits)
a. Oral anticoagulants (INR 2-4: rarely used in ICU)
i. Prosthetic valves (Mitral more than aortic valves)
ii. Previous thromboembolism

iii. Maintenance of thromboprophylaxis in high risk patients (fracture pelvis)

3. Protocol for DVT prophylaxis subcutaneous Enoxaparin
a. Routine monitoring is not necessary. If required, measure anti-Xa levels
b. Indications: Virtually all ICU patients
a. Previous history of DVT, embolism
b. Post joint replacement
c. Pelvic, lower limb fractures (consider high dose)
d. Prolonged femoral venous catheters
e. Prolonged immobility, neuromuscular wasting (for example, Guillain Barre, Polyneuropathy, Spinal injury)

c. Contraindications
i. Young, short stay patients (less than 24 hours) for example, overdoses
ii. Post neurosurgery / eye surgery
iii. Acute head injury with parenchyma lesions
iv. Intracranial hemorrhage
v. Active bleeding, Coagulopathy

vi. Previous documented HITS

NB: Consult with appropriate speciality if unclear when safe to start Enoxaparin (For example, following head injury)

d. In patients who cannot receive subcutaneous Heparin
i. TED stockings
ii. Calf stimulators
iii. Consider temporary IVC filter in high-risk patients

4. Protocol for Heparin induced Thrombocytopaenia Syndrome (HITS)
a. General Principles
i. Occurs in 1-5% patients: possible increase in incidence with CVVHDF
ii. Severe from

- 7-10 days after starting Heparin

- Autoimmune mediated platelet aggregation with thrombosis
- Not dose related: both with high dose and, in sensitized patients, low dose heparin (including vascular catheter flushes)
- Mild form: early onset, often less severe and dose related

b. Suspected HITS: unexplained thrombocytopaenia and / or thrombosis in a patient receiving Heparin

i. Consider other causes of thrombocytopaenia
ii. With hold all Heparin
iii. HITS screen: 10 ml blood in blue top container (includes LMWH across reactivity)

c. Confirmed HITS
i. Cease all Heparin (including flushing bags)
ii. Consider indications for Heparinisation of use other agent, for example, Danaparoid
iii. Document Heparin allergy in case notes

iv. Cross reactivity with low molecular weight Heparin test:
None
1. Start Enoxaparin
2. Monitor the platelet count (*test is not 100% sensitive)
b. Positive: anticoagulation indicated
1. Life threatening thrombosis/embolism
i. Start Danaparoid
ii. Consider a Caval filter

2. Short-term anticoagulation for example, patency of CVVHDF circuit: consider no anticoagulants or IV Prostacyclin

3. Prophylaxis: consider TED stockings or subcutaneous Danaparoid

TABLE 17

Drug	**Infusion / Dose**
Warfarin	1. Variable dose INR 2. Daily INR
Heparin (infusion)	1. 25000u/50ml = 500 u/ml 2. Start at 2ml/hr (1000u/hr): titrate against APTT: 50-80s
® (sucut)	1. Prophylaxis: 20mg subcut daily if Creatitine clearance less than 30 ml / min 2. Treatment: 1mg/kg subcut bd-lean body mass, 1mg/kg subcut once daily if Creatitine clearance is less than 30ml/hr
Prostacyclin (infusion)	1. Dose: 0.2-0.6 g/kg/hr 2. 500 g (+10ml diluent): add to 40ml NS = 10 g/ml solution 3. Start at 2ml/hr and monitor platelet count 4. May cause hypotesion

G. Renal drugs

1. General principles for use of diuretics in ICU

a) Oliguria in acutely ill patients is frequently a manifestation of hypovolaemia, decreased cardiac output, direct renal toxicity, or a combination of these factors. Therapy shall be directed toward these factors prior to the administration of a diuretic agent

b) Urine output, in the absence of diuretic use, represents one of the best markers of end-organ perfusion and is a useful guide to clinical management

2. Indication

a) Symptomatic fluid overload
b) Pulmonary fluid overload
   i. Congestive cardiac failure: Cor Pulmonale
c) Diuretic dependent Renal function
d) Hyperaldosterone states: ascites

3. Contraindications

- Hypovolaemic and / or Sodium depleted states
- Known drug hypersensitivity (especially Sulphonamide group)

4. Complications

a) Hypovolaemia
b) Hyperosmolar states due to inappropriate dieresis in hypovolaemia
c) Potentiation of renal failure secondary to hypovolaemia
d) Electrolyte disturbance especially decreased Potassium, Magnesium, Phosphate, metabolic alkalosis
e) Naturesis and Kaliuresis shall alter urine electrolytes and osmolality for 24-48 hours post due

TABLE: 22

Drug	Dose	Indications
Frusemide	40-250mg IV / oral prn / bd	1. First line, potent loop diuretic 2. Doses may be increased in diuretic dependence 3. K+, MG, Po4, metabolic alkalosis
Acetazolamide	250-500 mg IV	1. Carbonic anhydrate inhibitor : alkaline diuresis with HCO3 – excretion 2. Used for severe metabolic alkalosis after correction of hypovolaemia : K+, Mg, PO4

WEIGHT BASED HEPARIN DOSING

INITIAL THERAPY

Bolus 60-80 units / kg
Infusion 10-15 units / kg / hr

ADJUSTMENT

aPTT < 40 80 Units/Kg Iv bolus; increase infusion by 4 units/Kg/hr
aPPT < 40-54 40 Units/Kg Iv bolus; increase infusion by 2 units/Kg/hr
aPPT 50-80 No Change
aPPT 81-99 Decrease infusion by 2 units/Kg/hr
aPTT > 99 Hold for 1 hr; decrease infusion by 3 units/Kg/hr

a. Round all doses to nearest 100 units
b. Maximum 12,000 units
c. Maximum, 2,000 units/hr
d. Draw a PTT 6 hrs after any bolus or change in infusion rate

FLUIDS AND ELECTROLYTES

PRINCIPLES OF FLUID MANAGEMENT

1. All fluids, infusions are reviewed daily and rewritten

2. Assessment of volume status and fluid balance involves all of the following

a. Clinical markers
   i. Skin turgor, mucous membranes
   ii. Pulse, blood pressure
   iii. Peripheral perfusion, capillary refill
   iv. RAP, PAOP
   v. CxR

b. Biochemical markers
   i. Serum Sodium, Chlorides, osmolality
   ii. Urea/Creatinine (ratio)
   iii. Bicarbonate
   iv. Haematocrit

c. Charted fluid balance
   i. Total intake including drug/infusion volumes
   ii. Total output including urine output, NG losses, blood loss
   iii. Insensible losses due to pyrexia, transcellular shifts, ect

3. Fluids shall be considered in two components

a. Maintenance fluids

   i. Usually crystalloids, (according to Sodium)
   a. Lactate ringer

b. 5% Dextrose
c. Normal saline

ii. Usual volumes: 30-40 ml/kg/day at 80-120ml/hr
iii. TPN

b. Replacement / resuscitation fluids

i. Usually colloids:
a. Albumin 20%, or

b. Gelofusin
ii. Blood and component therapy as indicated
iii. Crystalloid replacement is usually used for excessive Renal, Enteric and burns losses

TOTAL PARENTERAL NUTRITION POLICY

Definition

Total parenteral nutrition (TPN) is a way of supplying all the nutritional needs of the body by bypassing the digestive system and dripping nutrient solution directly into a vein

Desired Outcome

The provision of adequate nutrition and prevention of malnutrition in a patient who is unable to be fed enterally

INDICATIONS AND CONTRAINDICATIONS

Indications

1. Patients with an inability to absorb nutrients via the gastrointestinal tract. This includes severe malabsorption, short bowel syndrome, intractable vomiting or diarrhea, and radiation enteritis, etc
2. Patients with severe acute pancreatitis requiring bowel rest
3. Severe malnutrition or catabolism when the gastrointestinal tract is not usable within 5 days
4. Patients where adequate enteral nutrient intake cannot be established within 7 to 10 days
5. Patients without ability to be fed by tubefeeding who will benefit from preoperative nutrition support to boost immune function

Contraindications

1. Patients who have a functional and usable gastrointestinal tract capable of absorption of adequate nutrients
2. Treatment anticipated for less then 5 days in patients without severe malnutrition

3. When the risks of parenteral nutrition is judged to exceed potential benefits
4. Prognosis that does not warrant aggressive nutrition support, and when such action is not desired by the patients or legal guardians in accordance with hospital policy

PERIOPERATIVE NUTRITION

The American Society for Parenteral and Enteral Nutrition recommends that perioperative nutrition support be provided to malnourished patients who require non-emergent surgery as well as to those who are expected to undergo a period substantial preoperative starvation. Patients are considered malnourished if they have inadequate nutrient intake for greater than 7 days, or if they have a weight loss greater then 10% of their pre-illness body weight. A preoperative nutrition support regimen of 7-10days will allow for a boost to the immune system, not for weight gain. Patients should be considered for enteral tube feedings first, if possible. Enteral tube feedings and parenteral

nutrition can be given simultaneously if enteral feedings cannot provide sufficient nutrients alone

PERIPHERAL PARENTERAL NUTRITION (PPN) is indicated for short term use in patients who are likely to regain gastrointestinal function within 7-10 days. Patients with poor venous access, high nutrient requirements, or severe fluid restriction or poor peripheral veins are Osmolarity is the limiting factor for PPN solutions. Osmolarity should not exceed 600-900mOsm/liter

PPN does not have the risks associated with central or total parenteral nutrition, but there are still some concerns to be considered. Thrombophlebitis is the most common occurrence. This complication can be limited by only using PPN with a low osmolarity. Infiltration, catheter embolism, and sepsis have also been known to occur with the use of PPN

CENTRAL OR TOTAL PARENTERAL NUTRITION (CPN OR TPN) is indicated for patients requiring nutrition support for greater than 7-10 days. TPN is used for patients with high kcalorie needs, severe fluid restriction, or poor peripheral access. It requires central venous access to supply dextrose concentrations greater than 10% and amino acid concentrations greater than 3.5%. Infusion into a large diameter vein dilutes hypertonic solutions quickly. Permanent or tunneled catheters can be placed for long term therapy

Steps:

1) TPN is prescribed by specialist medical officer only. Pharmacy must be notified as soon as possible to supply bags
2) If not already present, insertion of central venous line (includes Hickmann's lines or PICC lines, as appropriate) is arranged. The subclavian route is preferred if the line is to be specifically inserted for TPN. All central lines must have position confirmed on X-ray before use
3) The TRIPLE PHASE TPN/DOUBLE PHASE bag is prepared by rolling (to break the baffles and ensure adequate mixing of all chambers) and the giving set is prepared. This attached to a DEDICATED lumen of the central

line. Once mixed a bag can only hang for 24 hours and must then be changed

4) Infusion is commenced at 50 ml/hr, for 24 hours. When stable this is increased to 80ml/hr, which is the usual adult daily dose (corresponds to 2 litres per day). Lower starting rates may be required if there are electrolyte abnormalities. Higher daily rates may be required in certain patients after consultation – maximum daily dose is 36 ml/kg/day
5) Blood sugar level (finger prick) must be monitored every 4 hours for the first 48 hours, then daily if stable. If blood sugar > 200mg/dl then commence insulin infusion and titrate

Monitoring:

CBC, electrolytes, and BUN should be monitored daily for inpatients Blood glucose should be monitored q 4 until stable. Fluid intake and output should be monitored continuously. When the patient becomes stable, blood tests can be done much less often.

Liver function tests should be done. Plasma proteins (eg, serum albumin), prothrombin time; plasma and urine osmolality; and Ca, Mg, and phosphate (not during glucose infusion) should be measured weekly

NURSING CONSIDERATION FOR TPN

Administer TPN solution at constant rate
Change the tubing and filter if the system shows. Flushing or irrigation of the system shall be avoided
Assess and document vital signs, glucose monitoring, I&O, and routine weights accurately. This is essential to monitoring effectiveness of TPN Therapy
Observe for skin rashes, flushing, color changes, or other adverse or allergic type reactions

Notify the physician immediately is sepsis is suspected

TPN solutions hung, but not infused, shall be discarded after 24 hours
During the course of TPN therapy, if the solution is not readily available from the
Pharmacy, the nurse must contact the physician for an appropriate order
Only Registered Nurses are allowed to hang TPN

COMPLICATIONS

MECHANICAL:

Pneumothorax
Hemothorax
Intravascular Misplacement – often IJV
Catheter Embolism – sheared tip
Air Embolish
Venous Thrombosis

METABOLIC:

Hyperglycemia
CO2 Retention
Hyperammonemia and Hepatic Encephalopathy
Hyperlipidemia
Elevated liver function tests
Fluid and Electrolyte Disorders
Sepsis

ELECTROLYTES MANAGEMENT

1. General Principles

a. Total body water (60% total body weight):

i. Intracellular fluid: predominant ions: K+, PO4
ii. Extracellular fluid:
b. 75% interstitial fluid: predominant ions: Na+, Cl-
c. 25% plasma volume (PV)

d. Osmotic equilibrium is maintained by Na+/K+ pump
i. Extra cellular fluid ions therefore reflect total osmolality
ii. Magnesium is a cofactor for this pump

c. Most electrolyte disturbances in critically ill patients relate to changes in the distribution and concentrations of the predominant ECF and ICF ions

d. As a general rule, changes in one ion shall be reflected in he associated cation or anion

e. Electrolyte disturbances shall be considered in terms of the following groups:
i. Erroneous results
a. Lab error
b. Bloods taken from a drip arm
c. Haemolysed specimen – traumatic (old IA lines), delayed samples
ii. Decreased or increased intake
iii. Decreased or increased losses: usually
a. Renal
b. Extra renal: GIT, skin losses
iv. Transceullar shifts:

f. Treatment shall be directed at the underlying cause.
g. Rapid correction of electrolytes disturbances may be deleterious
h. The following paragraphs outline the common electrolyte disturbances

2. Hyponatraemia: Sodium less than 130 mmol/l
a. Aetiology / classification

i. Hyperosmolar: measured osmolality more than 290
a. Hyperglycaemia
b. Mannitol
c. Ethanol, Methanol, Ethylene, Glycol
ii. Isoosmolal: measures osmolality 270-290

a. Hyperlipidaemia
b. Hyperprteinaemia not seen with ion-specific electrodes
c. IVT arm sample

iii. Hypoosmolal: measured osmolality is less then 270

a. Hypovolaemia (Sodium depletion)

1. Renal
i. Diuretics
ii. Addison's
iii. Polyuric Renal failure

2. Extra renal
i. GIT
ii. Burns

3. Transcellular water loss

b. Hypervolaemia (Water excess)

1. Extra renal
i. Excessive intake (IV 5% Dextrose)
ii. Oedematous states – CCF, cirrhosis, Nephrotic syndrome decreased Albumin

2. Renal
i. Acute Renal polydipsia

c. Normovolaemia

1. Psychogenic polydipsia
2. SIADH
3. Hypothyroidism

b. Diagnosis and management

i. Iso and Hyper-osmolal
a. Factitious ignore and manage underlying condition
b. Hyperglycaemia: (BSL 10 mmol/l Na+ 3 mmol/l)

1. Hyponatraemia per se, is real, but treatment is directed at the underlying cause, where correction of the hyperglycaemia shall correct the plasma (Na) as shown
2. NB: Total body sodium deficit may co-exist with hyperglycaemia / Diabetic Ketoacidosis

b. Mannitol
1. Decreased Sodium early, then diuresis and late increased. Sodium are more problematic
2. Maintain adequate plasma volume with Normal Saline initially

c. Alcohols: permeate solutes, therefore (Na) less problematic

ii. Hypovolaemic states
a. Restore volume with colloid or Normal Saline according to clinical markers: urine output, plasma (Na), RAP
b. Aim for slow sodium correction: 2 mmol/l/hr, unless seizures
c. Urine Sodium is un-interpretable after diuretics or Catecholamines for 24 hours after dose

iii. Hypervolaemic states: *most common clinically
a. Fluid restriction less than 15 ml/kg/day

1. "Water Excess" ~ (140 – Na+)/140 x (Body Wt x 0.6) for example, 70 kg patient with plasma (Na) = 120 mmol/l = (140 -120)/ 140 x (70 kg x 0.6) = 6 litres

2. Shall slowly correct excess – ADH group and "reset osmostat"

3. Treat the underlying cause – CCF, Nephrotic Syndrome, ascites

iv. SIADH

a. Diagnosis

1. Hypo-osmolar Hyponatraemia
2. Urine osmolality plasma osmolality
3. Urine Sodium is greater than 40 mosm/l
4. Normal endocrine, Renal, Hepatic, Cardiac Function, No diuretics
5. Corrected by fluid restriction alone

b. Management: fluid restriction

v. Severe Symptomatic Hyponatraemia: fitting, or decreased consciousness

a. Resusciation / ABC
b. Hypertonic saline (3, 20 and 29%) is rarely indicated; always discuss use with the dealing Consultant
c. Correct (Na+) rapidly only to 120mmol/l
d. Thereafter, slow correction with Normal saline over 24-36 hours (2 mmol/l/hr)
e. Treat the underlying cause

3. Hypernatraemia: Sodium more than 145 mmol/I

a. Aetiology / Classification: always hyperosmolar

i. Hypovolaemic H2O loss > Na+ (most body fluids have a (Na+) < plasma net water loss)

1. Renal

1. Diuretics, glucosuria

2. 2.ARF/CRF, partial obstruction
3. 3.Diabetes Insipidus

b. GIT losses: Diarrhoea, vomiting, fistulae, small bowel obstruction

c. Respiratory – Intermittent positive pressure ventilation with dry gases

d. Skin losses

1. Fever, high ambient temperature
2. Thyrotoxicosis
3. Vasodilatory states
4. Exfoliative skin disorders, burns

ii.Iso-hypovolaemic "pure water" less (plasma osmotic pressure, tends to maintain intravascular volume mild-moderate decrease in ECF and ICF)
iii. Iso-hyervolaemic Na+ gain > H2O gain (Plasma osmality ADH secretion ECFV, with subsequent renal escape, thus Oedema in this group is rare)

a. Iatrogenic (major cause): NaHCO3, feeding formulate, TPN
b. Mineralocorticoid excess: usually has 1-3 of excess total body weight
1. Conn's, Cushing's syndromes
2. Steriod excess

b. Management
i. Hypovolaemic states
a. Restore volume according to clinical markers: BP, HR, urine output, RAP
1. Colliod: initial resusciatation, severe Hypovolaemic states
2. Hartman's solution – slightly hypo-osmolar

HYPERTONIC SALINE 3%

Classification

Electrolyte

Alternate Name

Hypertonic saline 3%, NaCl 3%

Indications

- Management of severe sodium chloride depletion when rapid electrolyte restoration is essential
- Management of sever symptoms of hyponatremia including seizures, coma, and focal nuerologic signs

Reconstitution and Stability

- Stable at room temperature

Compatibility

- Compatible with commonly used IV solutions
- Do not mix with other medications

Routes of Administration

- IV intermittent – maximum rate of 100 ml / hr
- IV infusion – maximum rate of 100 ml / hr

Administration Policy

H – The IV infusion must be controlled by an automated infusion control device

Dosage

The following formula can be used to calculate sodium deficit:

Sodium deficit (mmol) = (desired – current serum sodium, mmol/L) x (total body water, L) = (140 mmol/L – patient's serum sodium) x (0.6 L/kg x patient's weight, kg

Using NaCl 3% for replacement, administer one-third to one-half the calculated sodium deficit over the first 8-12 hours at rate of 25-50 mL/hour, not to exceed a maximum rate of 100 mL/hour. There should be no attempt to normalize serum sodium levels in the first 24 hours

Monitor serum sodium and electrolytes, input and output, and vitals signs closely to assess need for additional sodium chloride 3% (every 1-4 hours initially)

Continue treatment until a serum sodium of 120-125 mmol/L or neurologic symptoms improve; remainder of deficit can be replaced over severe days

Once serum sodium greater than 120-125 mmol/L, use NaCl 0.9% (normal saline) to correct the additional deficit over 3-5 days

A loop diuretic (e.g. furosemide) may be added to prevent sodium overload and enhance free-water excretion

Potential Hazards of Parenteral Adminitration

- Thrombophlebitis
- Electrolytes, volume and acid-base disturbances
- Congestive Heart Failure and Pulmonary Edema

IMPORTANT IMPLICATIONS

- Fluid balance, serum electrolyte concentrations (sodium, potassium, bicarbonate, chloride, magnesium), and acid-base balance should be monitored closely
- Use with caution if congestive heart failure, liver cirrhosis, severe renal failure, urinary tract obstruction, or in patients receiving drugs that can cause sodium retention, such as corticosteroids

- Sodium chloride 3% contains 513 mmol/L each of sodium and chloride with a calculated osmolarity of 1025 mOsm/L

1. Hypokalaemia: Potassium less than 3.0 mmol/l

a. Etiology / Classification

i. Transcellular shift

1. Alkalemia pH ~ 0.1 {K+} pl~0.5 mmol/l
2. Catecholamines / Salbutamol
3. Insulin
4. Familial periodic paralysis
5. Anabolism – refeeding effect
6. Hypomagnesaemia – ICF Potassium depletion

ii. Reduced intake

a. Starvation
b. TPN

iii. Increased clearance

a. Renal

1. Diuretics leading to raised distal tubular flow
   i. PT agents – Acetazolamide, Mannitol
   ii. Loop agents – Frusemide, Bemutanide
   iii. Early DT – Thiazides
2. Steriods / Mineralocorticoid excess

3. Anionic drugs
    i. Antibiotics (Pencillins)
    ii. Amphotericin
4. Hypomagnesaemia, Hypocalcaemia
5. Lithium
6. RTA I, II

b. Non-renal

1. GIT
    i. Villous Adenoma
    ii. Ureterosigmoidostomy
    iii. Fistulae, malabsortion syndromes

2. Skin losses

b. Management

i. Treat underlying cause
ii. Correct hypovolaemia: volume contraction shall potentiate both alkalosis and hypokalemia
iii. Always add Magnesium: normomagnesaemia is essential for correction of hypokalaemia
iv. Causes and treatment of hypophosphataemia same as decreased Potassium
v. Potassium preparations
   a. KCL: 10 ml = 1g = 13.4 mmol/l
   b. KH2PO4 : 10 ml = 10 mmol/l
   c. K-acetate : 10 ml at 5 mmol/ml 50 mmol K+ +50 mmol acetate
   d. K-acetate : 10 ml at 5 mmol/ml 50 mmol K+ +50 mmol acetate (Bicarbonate)

Potassium Infusion Guidelines

Standard concentrations and infusion rates will be utilized for replacement infusions of potassium chloride

Procedure

If a patient requires more potassium than the maximum allowed per bag, consecutive bags may be infused. At no time will an individual bag be dispensed with greater than the maximum concentration

A central line is preferred for potassium infusions

All potassium infusions should be administrated via an infusion pump

Solutions containing potassium will be dispensed/verified by the pharmacist. It is the nurse's responsibility to ensure appropriate administration and monitoring (i.e. central line along with vital signs and rhythm documentation every hour if administering Potassium Chloride at a rate of 20 mEq/hr to a patient)

Potassium Chloride Infusions

A. Adult Patients

a. Standard and maxium concentration of KCL for replacement infusions: 10mEq per 50 mL. Potassium may be further diluted in large volumes of fluid to alleviate injection site irritation

b. Maximum concentration for maintenance fluids is 60mEq per Liter. Parenteral nutrition admixtures are an exception to this requirement

B. Pediatric Patients

a. Standard and maximum concentration of KCL for replacement infusion: 10mEq per 50 mL. Potassium may be further diluted in larger volumes of fluid to alleviate injection site irritation

b. Maximum concentration for maintenance fluids is 80mEq per Liter provided that maximum infusion rate is not exceeded and it is infused via a central line. In select hematology/oncology patients, the maximum concentration of potassium per liter may exceed 80 mEq per Liter with Attending physician approval only. Parenteral nutrition admixtures are an exception to this requirement

VI. Potassium Chloride Infusion Rates for Replacement

A. Adult Patients

a. Acute patient care setting may infuse at a rate of 10 mEq of potassium per hour
b. Patients in Intensive Care Units may receive potassium chloride infusions at a rate of 20 mEq of potassium per hour when the patient is on a cardiac monitor. A central line is required for administration
c. Select ICU patients with a central line may receive potassium chloride infusions at a rate not a exceed 40 mEq per hour with Attending Physician approval only

B. Pediatric Patients

a. Maximum infusion rate is 0.3 mEq/kg/hour (maximum 10 mEq per hour) if the patient is not being monitored by EKG

b. Potassium chloride infusion rate may be increased to a rate of 0.5mEq/kg/hr (maximum 10mEq per hour) on patients without a cardiac monitor with attending Physician approval only
c. Patient in Intensive Care Units may receive potassium chloride infusions at a rate not to exceed 1 mEq/kg/hour (maximum 20 mEq per hour) when the patient is on a cardiac monitor. A central line is required for administration

Potassium Infusion Guidelines

ADULT	PEDIATRIC		NEONATE
POTASSIUM CHLORIDE			
Standard Concentration for replacement infusions	10mEq per 50 ml	10mEq per 50 ml	0.2 mEq/ml
Maximum Concentration for maintenance infusions	10mEq per litre	10mEq per litre[central line only]	6mEq/kg/day;0.2 mEq/ml
Exceptions	Parenteral nutrition	Parenteral nutrition;Select pediatric hematology/oncology patients with attending approval only	>6mEq/kg/day requires attending approval
Standard infusion rates for replacement			

Non-critical care patients (no cardiac monitor)	10 m Eq/hr	< 0.3 mEq/kg/hr	< 0.3 mEq/kg/hr
Critical care patients on a cardiac monitor	10 m Eq/hr	< 0.5 mEq/kg/hr	< 0.5 mEq/kg/hr
Maximum Infusion Rates			
Non-critical care patients (no cardiac monitor)	10 m Eq/hr	0.3 mEq/kg/hr (up to 0.5 mEq/kg/hr with attending approval only)	0.3 mEq/kg/hr
Intensive care patients on a cardiac monitor	20 m Eq/hr (Central line only)	1 mEq/kg/hr (maximum 20 mEq/hr) (central line only)	1 mEq/kg/hr

5.Hyperkalaemia : Potassium more than 5.0mmol/1

a. Aetiology/Classification

i) Artefactual

- Drip arm Specimen
- Tourniquet/Haemolysed(extravascular)

c. Thrombocytosis (more than 750,000)

d. Leucocytosis more than 50,000

ii. Transcellular exchange

a. Acidosis breakdown

b. Tissue breakdown

c. Rhabdomyolysis, haemolysis (intravascular), ischemia / reperfusion

d. Severe burns
e. Tumour Lysis Syndrome
f. Leukaemia
g. Familial periodic paralysis
h. Suxamethonium paralysis
i. Insulin deficiency: DKA

NB: Normo-or hypokalaemia in the presence of severe DKA is associated with a marked total body potassium deficit, which shall be addressed prior to correction of the acidaemia

j. Increased ECF tonicity: movement of water from cells the (K+) ICF and passive diffusion seen with large doses of Mannitol given rapidly (1.5-2.0 g/kg), Hyperkalaemia of DKA is due to this addition to the acidaemia and Insulin deficiency
k. Beta blockers

iii. Increased intake-rarely a problem unless impaired Renal function

a. Direct IV / oral
b. Drugs (Penicillins)
c. Transfusion

iv. Reduced clearance

a. Acute Renal failure

1. Any cause for decreased distal tubular flow, or decreased distal NaCl Delivery

2. Hypoaldosteronium (Mineralocorticoid deficiency, Addison's) – K+ is multifactorial (K+ ICF K+ ECF, distal flow and DT Aldosterone effect)

a. Type IV RTA
b. Potassium sparing diuretics
c. Aldosterone antagonists – Spironolactone

3. Distal Sodium channel inhibitors – Amiloride, Triamterene

a. Management
i. The clinical scenario shall dictate treatment
ii. Acute Potassium levels more than 6.0 mmol/ is a medical emergency
iii. Associated with acute ECG changes, or haemodynamic compromise in following order
b. Calcium Chloride 10 ml IV stat
c. Sodium Bicarbonate 50 ml IV stat
d. Glucose 50% 50 ml and Insulin 10 units
e. Salbutamol nebulisation continuously
iv. Refractory or persistent

a. CVVHDF
b. Intermittent dialysis
v. Chronic increase in Potassium levels or slow rate of rise or no ECG changes:
Resonium 30 g oral / PR 8 hourly
vi. Address aetiological factors
viii. Normalise renal function / volume status

E. Other Electrolyte Abnormalities

1. Calcium

Calcium is required for muscle contraction, nerve impulse transmission, hormone secretion, blood clotting, cell division, cell motility, and wound healing. Effective calcium levels are best assessed by using ionized calcium measurements, if available. If treatment decisions are based on total serum calcium, the albumin concentration must be considered. In general, for each increase or decrease in serum albumin of 1 g/dL, the serum calcium increases or decreases by 0.8 mg/dL (0.2 mmol/L). However, the relationship between albumin and serum calcium is less reliable in crtitically ill patients

a. Hypocalcemia (Total Calcium < 8.5mg/dL {< 2.12 mmol/L}, Ionized Calcium < 1.0

Hypocalcemia is common in critically ill patients and results from impairment of the parathyroid and / or vitamin D systems (Table. 12-5). Cardiovascular abnormalities, the most common clinical manifestations of hypocalcemia in critically ill patients, include hypotension, bradycardia, arrhythmias, heart failure, cardiac arrest, digitialis insensivity, and QT inverval and ST segment prolongation. Neuromuscular manifestations include weakness, muscle spasm, laryngospam, hyperreflexia, seizures, tetany, and paresthesias

Table 19: Causes of hypocalcemia

Hypoparathyroidism	Malabsorption
Sepsis	Liver disease
Burns	Renal disease
Rhabdomyolysis	Calcium chelators
Pancreatitis	Hypomagnesemia
	Massive transfusion

Treatment is aimed at correcting the underlying disease process and any concomitant electrolyte abnormalities, and administering calcium. Mild

hypocalcemia is well tolerated, and aggressive treatment may result in tissue injury (especially during Ischemic and septic states). If the hypocalcemia is severe or if the patient is symptomatic, administer 100 mg calcium intravenously over 5 to 10 minutes (3 to 4 mL of 10% calcium chloride, 10mL of 10% calcium gluconate), followed by 0.3 to 2.0 mg/kg/hr. Calcium preparations vary in the content of calcium: 10% calcium chloride 1g = 10 mL = 272 mg of calcium; 10% calcium may be replaced via the Enteral rout (i.e., 500 to 1000 mg every 6 hours).

Monitor ionized or total calcium levels frequently during treatment, and adjust repletion to maintain calcium in the lower normal range, so as not to suppress parathyroid gland function. If calcium replacement alone fails to maintain the circulating calcium level, consider administration of vitamin D and magnesium. Adverse effects of calcium administration include Hypercalcemia, bradycardia, nausea/vomiting flushing, tissue calcium precipitation, and digitals toxicity

b. Hypercalcemia (Total Calcium > 11 mg/dl (>2.75 mmol/L), Ionized Calcium > 1.3 mmol/L)

The most common causes of Hypercalcemia are the result of calcium release from bone. The clinical manifestations of Hypercalcemia relate primarily to the cardiovascular and neuromuscular systems and include hypertension, cardiac ischemia, arrhythmias, bradycardia, conduction abnormalities, digitalis toxicity, dehydration, hypotension, weakness, depressed mentation, coma, seizures, and sudden death. Gastrointestinal manifestations include

nausea/vomiting, anorexia, abdominal pain, constipation, Pancreatitis, and ulcer disease. Nephrogenic diabetes insipidus with polyuria may occur and when present contributes to volume depletion. Renal stones, nephrocalcinosis, and renal failure are also encountered

Table. 20 Causes of Hypercalcemia

Hyperparathyroidism	Excess vitamin A or D intake
Malignancy	Thyrotoxicosis
Immobilization	Ganulomatous disease

Treatment of Hypercalcemia is aimed at controlling the underlying disease, rehydrating the patient, rehydrating the patient, and lowering the calcium level. The circulating calcium level frequently needs to be lowered while the primary disease is being evaluated and treated. Intravascular volume should be restored with normal saline to assure adequate tissue perfusion and renal blood flow (urine output 2 to 3 mL/kg/hr). Saline also decreases renal tubular calcium reabsorption. Diuresis with a loop diuretic further increases renal calcium loss. In patients with renal failure or life-threatening Hypercalcemia, calcium levels may be lowered with dialysis
After initial stabilization, therapy with calcitonin, mithrramycin, or diphosphonates can be considered

3. Hypophosphatemia (Phosphate < 2.5 mg/dl (0.81 mmol/L})

Phosphate is important in cellular energy metabolism. Hypophosphatemia results from transcellular shifts, renal loss, gastrointestinal loss, or inadequate intake. Phosphate depletion primarily affects the neuromuscular and central nervous systems. Clinical manifestations include muscle weakness, respiratory failure, Rhabdomyolysis, paresthesias, lethargy, disorientation, obtundation, coma, and seizures. Other manifestations include impaired renal tubular function, impaired pressor responses, hepatic

dysfunction, immune dysfunction, impaired protein synthesis, hemolysis, impaired platelet function, and impaired oxygen offloading from hemoglobin

Table 21: **Causes of hypophosphatemia**

Transcelluar Shift	**Renal Loss**	**Gastrointestinal Loss**	**Decreased Intake**
Acute alkalosis	Hypoparathyroidism	Malabsorption	Malnutrition
Carbohydrate administration	Diuretic use	Diarrhea	Parenteral nutrition
Drugs (insulin, epinephrine)	Hypokalemia	Intestinal fisulas	
	Hypomagnesemia	Antacids	
	Steroids		

Treatment of hypophosphaemia consists of controlling the underlying disease, removing offending drugs, correcting electrolyte abnormalities, and replacing phosphate. Phosphate levels < 1 mg/dl (<0.32 mmol/L) associated with symptoms are considered life-threatening and require Immediate treatment.

For emergency treatment, administer phosphate at 0.6 to 0.9 mg/kg/hr intravenously. When circulating phosphate levels are stable, maintenance replacement of phosphate is 1000 mg/day intravenously plus excess losses (i.e., in urine or stool). Phosphate may be administrated as potassium phosphate (93 mg phosphate/mL; 1.1 mmol/mL patient with serum phosphate levels > 1 to 1.5 mg/dL (> 0.32 to 0.48 mmol/mL).

Serum phosphate should be monitored during repletion and therapy adjusted to achieve a circulating level of 3 to 4 mg/dL (0.97 to 1.29 mmol/L). Adverse effects of phosphate administration include hyperphosphatemi, hypocalcemia, tissue calcium precipitation, renal injury, and diarrhea (enteral phosphate)

MAGNESIUM REPLACEMENT, MANAGEMENT OF THE PATIENT REQUIRING

General electrolytes replacement information:

- Normal magnesium levels are necessary in order to properly replete calcium and potassium. If the patient's clinical status allows, replace magnesium before calcium and potassium
- It is not necessary to recheck a calcium or potassium level after replacing magnesium and prior to replacing potassium. However, after repleting any necessary eletrolytes recheck lab values per respective protocol

Indications for use

Magnesium replacement is considered if the following conditions exits

Serum magnesium level less than or equal to 1.9 mEq/L

Serum creatitine < 2 mg/dl

Patient's hear rate is > 50 beats per minute and systolic BP > 90 mmHg
Absence of renal disease requiring renal replacement therapy or dialysis.
Absence of neuro mascular disease (i.e., mayasthenia gravis, guillan barre syndrome)
IV magnesium must be give via infusion pump.

Before starting the magnesium infusion assess the following patients parameters:

Vitals signs
Arrhythmia status
Serum magnesium
Serum creatitine
Post medical history for presence of neuromuscular disease

Asses IV site for patency before beginning infusion

Assess for sources of magnesium loss (i.e. diuresis, excessive vomiting, diarrhea, hypokalemia)

Symptoms of hypomagnesemia:

- Neuromuscular irritability (muscle tremors, weakness, fasciculation, tetancy))
- CNS: nystagmus, ataxia, vertigo, psychiatric abnormality
- CV tachycardia, prolonged PR and QT interval, abnormal T waves other hypokalemia

Symptoms of hypermagnesemia:

- CV: bradycardia, increased QRS, hypotension
- Neuromascular: mental confusion, drowsiness, paralysis, coma
- GI: N/V, diaphoresis
- Resp: paralysis of respiratory muscles with (i.e. respiratory arrest and flash pulmonary edema)

1.0 Interventions

1.1 Infuse via infusion pump at a rate no faster than 2 grams per hour (1gram/hr preferred)

1.2 Magnesium to be diluted as follows – 1 gram to be diluted in 50 ml of NS

Serum Mg level (mEq/L)	**Dose of Mg**	**Infusion time**	**Blood Draw after Infusion**
> 2	0 grams		
1.8-1.9	2 grams	1-2 hours	With next am labs
1.5-1.7	4 grams	2-4 hours	With next am labs
1.2-.14	6 grams	3-6 hours	12 hours
<1.2	6 grams Obtain STAT ECG and notify MD	3-6 hours or MD order	12 hours or per MD order

2.0 Consider expert help in case of;

Serum magnesium level less than 1.2 mEq/L (see above chart)
Serum magnesium level > 3 mRq/L
Signs and symptoms or hypo/hyper magnesemia as described in section 3.5 and 3.6

F. Metabolic disturbances

a. Acute Adrenal Insufficiency

Lack of specific signs and symptoms makes early recognition of acute adrenal insufficiency difficult. Adrenal insufficiency may result from failure of the adrenal glands (e.g., autoimmune disease, granulomatous disease, human

immunodeficiency virus (HIV) infection, adrenal hemorrhage, meningococcemia, ketoconazole) or failure of the hypothalamic/pituitary axis (e.g., withdrawal from glucocorticoid therapy). Primary adrenal gland failure

results in loss of both glucocorticoid and mineralocorticoid secretion, whereas secondary adrenal gland failure causes only glucocorticoid deficiency.

Clinical manifestations suggestive of acute adrenal insufficiency include weakness, nausea, vomiting, abdominal pain, orthostatic hypotension, hypotension refractory to volume or vasopressor agents, and fever. Suggestive laboratory findings include hyponatremia, hyperkalemia, acidosis, hypoglycemia, and prerenal azotemia

Emergent treatment is indicated in critically ill patients, even if the diagnosis is not established. High-risk patients include those with the acquired immune deficiency syndrome (AIDS), disseminated tuberculosis, sepsis, or acute anticoagulation, as well as postcoronary artery bypass grafting patients and patients from whom glucocorticoid therapy was withdrawn within the past
12 months. When dexamethasone is used initially for emergent steroid replacement, a short adrenocorticotropic hormone (ACTH) stimulation test can be performed for diagnosis after resuscitative therapy is instituted because this corticosteroid dose not interfere with serum cortisol assay. The treatment steps for acute adrenal insufficiency are as follows:

- Obtain baseline blood samples for cortisol, electrolytes, etc
- Infuse D5 normal saline to support blood pressure
- Administer dexamethasone 4mg intravenously, then 4 mg intravenously every 6 hours
- Perform short adrenocorticotropic hormone stimulation test if needed for diagnosis
- If the diagnosis of adrenal insufficiency is confirmed, hydrocortisone 100 mg intravenously every 8 hours can be administrated. Some
-

  physicians prefer administration of hydrocortisone as a continuous infusion, 300 mg over 24 hours

- Test precipitating conditions.

b. Hyperglycemic Syndromes

Serious metabolic complications of diabetes result from a relative or absolute lack of insulin coupled with increased production of counter regulatory hormones such as glucagons, catecholamines, and others. Life-threatening hyperglycemic syndromes include diabetic ketoacidosis (DKA) and hyperglycemic hypersmolar state (HHS)

Clinical manifestations result from hyperglycemia and excess ketone production. Hyperglycemia causes hyperosmolality, osmotic diuresis fluid and electrolyte loss, dehydration, and volume depletion. Ketones (DKA) cause acidosis and osmotic diuresis. Clinical features of
hyperglycemic syndromes include weakness, dehydration, polydipsia, altered mental status, coma, tachycardia, arrhythmias, hypotension, anorexia, nausea, vomiting, ileus, abdominal pain, hyperpnea, and fruity odor to the breath (DKA). Laboratory investigation reveals hyperglycemia, hyperosmolality (more common in HHS), glucosuria, ketanemia/ketonuria (DKA), anion gap metabolic acidosis (DKA), hypokalemia, hyphosphatemia, hypomagnesemia, leukocytosis, azotemia, and elevated amylase and creatine phosphokinase.

The goals of treatment of hyperglycemic syndromes are to restore the fluid and electrolyte balance, provide insulin, and identify precipitating factors (e.g. infection, stroke, myocardial infarction, pancreatitis, corticosteroids). Volume deficits correlate with the severity of hyperglycemia and are usually greater in HHS. Normal saline should be used to replenish intravascular volume and restore hemodynamic stability and renal perfusion (i.e., 1 liter in the first hour, then 250 to 500 mL/hr as need). After administration of 1 to 2 L of normal saline, fluids with less chloride (e.g., 0.45% saline) should be

used to avoid or minimize the development of hyperchloremic metabolic acidosis. The corrected serum sodium (measured sodium+ (1.6 x glucose/100) should also be used to guide fluid selection. Urine output should be maintained at 1 to 3 mL/kg/hr to ensure adequate tissue perfusion and clearance of glucose. Invasive hemodynamic monitoring (e.g., arterial catheter, pulmonary artery catheter) may be required in patients with underlying cardiovascular disease.

Priorities in initial resuscitation of diabetic ketoacidosis

1. Institute crystalloid resuscitation, initially with normal saline
2. Institute insulin bolus (0.1 to 0.15 U/kg) and infusion at 0.1 U/kg/hr
3. Consider bicarbonate only if pH < 7.0
4. Look for precipitating cause of DKA (infection, myocardial infarction, pancreatitis, trauma, etc)
5. Add potassium chloride to IV fluids when serum potassium is known or expected to be low or normal, and urine output is documented
6. Add glucose to crystalloid infusion when serum glucose is 250 to 300 mg/dL (13.9 to 16.7 mmol/L). Do not decrease insulin infusion rate. Administer 10% dextrose if necessary to maintain serum glucose > 150 mg/dL (> 8.3 mmol/L)
7. Continue insulin infusion until ketosis is cleared (negative serum ketones with correction of increased anion gap)

Give 0.1 to 0.15 U/kg regular human insulin intravenously as a loading dose in DKA, followed by 5 to 10 units of regular insulin per hour (0.1 U/kg/hr). The intravenous rate is the most reliable and easiest to titrate. Because of the short half-life of intravenous insulin, a continuous infusion is necessary with serial monitoring of the blood glucose and electrolyte concentrations. Smaller doses of insulin are usually adequate in HHS. Monitor glucose levels frequently, and when glucose decreases to 250 to 300 mg/dL (13.9 to 16.7 mmol/L), switch to glucose-containing fluids to avoid hypoglycemia. Glucose-containing fluids may be started earlier if blood glucose cannot be monitored frequently. Administration of 10% dextrose may be necessary to maintain glucose levels > 150mg/dl (> 8.3 mmol/L) while continuing insulin infusion

for clearing ketones. When blood glucose is controlled, ketonemia has cleared, and the patient is stable, the patient may be advanced to subcutaneous insulin. An insulin sliding scale with subcutaneous administration of regular and longer-acting preparations can be started and should be overlap with discontinuation of the insulin infusion.

Insulin and correction of acidosis shift potassium intracellularly and may be lead precipitous drops in serum potassium levels. Potassium deficits range from 3 to 10 mmol/kg. Potassium should be added to fluid therapy as soon as serum potassium is recognized to be < 5.5 mmol/L and urine output is documented. Oral potassium replacement can be considered if nausea and vomiting are not present. Potassium and other electrolyte levels should be monitored frequently until levels stabilize and acidosis is resolved (eg.DKA)

Consider phosphate replacement if serum levels are low. Magnesium should be administration if hypomagnesemia is present. Acidosis is well tolerated by patients with DKA, and bicarbonate therapy, which is controversial, is rarely needed. Bicarbonate over 1 hour to increase pH > 7.0). Do not attempt to normalize blood pH with bicarbonate, since acidosis resolves as ketones are metabolized

G. Additional Metabolic Disturbances

1. Thyroid Storm

Thyroid storm usually occurs in patients with Graves' disease or toxic nodular goiter. Precipitating factors include infection, surgery labor/delivery, acute medical illness, trauma, or emotional stress. Thyroid storm is characterized by exaggerated classic manifestations of hyperthyroidism plus fever, CNS dysfunction (apathy, agitation, coma etc), congestive heart failure, arrhythmias, and gastrointestinal symptoms (Vomiting, diarrhea, and jaundice).

Therapy should be initiated immediately without awaiting results of thyroid hormone tests. An endocrinologist or critical care specialist should be

consulted as soon as possible. Supprotive and specific measure are listed below

Supportive measures:

- Cooling: acetaminophen, cooling blankets (salicylates should be avoided since salicylates affect binding of T4 to T4 to their binding proteins)
- Hydration: glucose-containing fluids
  Treat the precipitating cause
- Inhibit T4 synthesis: propylthiouracil 800 to 1200 mg orally initially, then 200 to 300 mg orally every 6 hours; or methimazole 80 to 120 mg orally or rectally initially, then 20 to 30 mg orally or rectally every 6 hours
- Inhibit adrenergic effects: Propranolol 1.0 to 10 mg intravenously (titrated) or 40 to 120 mg orally every 6 hours
- Inhibit T4 release: saturated solution of potassium iodide (SSKI) 5 drops orally every 6 to 8 hours; or sodium iodine 0.5 g intravenously every 12 hours. Initiate 1 hour after prophylthiouracil or methimazole is given
- Inhibit T4 to T3 conversion: dexamethasone 2 mg intravenously or orally every 6 hours; sodium ipodate 0.5 to 1.0 g orally every day.

Thyroid storm is infrequent in children. Occasionally, thyroid is seen in neonates born to mothers with hypothyroidism or circulating thyroid-stimulating antibodies. The thyroid disease spontaneously remits in 1 to 2 months, but treatment as listed below may be necessary during the period:

- Propylthiouracil 6 to 10mg/kg/day orally every 8 hours in equal doses or methimazole 0.6 to 0.7 mg/kg/day orally every 8 hours
- SSKI 0.3 mL orally every 8 hours
- Dexamethasone 0.2 mg/kg/day

- Propranolol 30 to 100µg/kg/day intravenously (titrated), then 0.5 to 10 mg/kg/day in three to four equal doses

2. Myxedema Coma

Myxedema coma (life-threatening hypothyroidism) is a rare manifestation of chronic hypothyroidism that occurs most frequently in elderly patients during winter months. Precipitating factors include infection, drugs (i.e. sedatives, hypnotics), surgery, neurologic disorder (e.g., Cerebrovascular accident, seizures), acute medical illness, trauma, and exposure to cold. Clinical manifestations are an exaggeration of less severe hypothyroidism and result in widespread organ dysfunction. Features of life-threatening hypothyroidism include hypothermia, obtundation/coma, bradycardia, heart failure, hypoventilation, hyporeflexia, hyponatremia, hypoglycemia, anemia, and decreased ECG voltage.

The diagnosis of myxedema coma is clinical, and treatment should be delayed pending laboratory results. Treatment with T4 is unlikely to cause harm if the diagnosis is in error, but delay in therapy can be fatal. An endocrinologist or clinical care specialist should be consulted as soon as possible. In addition to obtaining blood for thyroid hormone tests, the following measures should be instituted:

- Secure airway and ventilate, if necessary
- Treat hypotension with fluids
- Treat precipitating cause
- Provide passive warming (e.g., blankets)
- Provide glucose
- Treat significant hyponatremia with diuresis (e.g., loop diuretic)
- Give hydrocortisone 100 mg intravenously every 8 hours, or assess adrenal functions
- Administer L-thyroxine (T4), 300 to 500 µg daily; children, 4µg daily

H. Acid base disturbances in intensive care

a. Acid base disturbances in Intensive Care are frequently mixed metabolic and respiratory disorders.

b. Correction to these disturbances shall be directed at the underlying cause

and maintenance of cardiopulmonary homeostasis

c. Primary correction of an acid base disturbance with acid or alkali is seldom required

d. "Rules of thumb" *these are approximations only

i. Primary metabolic disturbances: last 2 drugs of pH shall reflect PaCO2

a. Met acid to minimum 7.10 for example, pH 7.25 PaCO2 mmHg

b. Met alkalosis to maximum 7.60 for example, pH 7.57 PaCO2 57 mmHg

ii. Primary respiratory acidosis

a. Raised Bicarbonate – 1mmol/lper 10mmHg PaCO2 below 40 to min 18

iii. Primary respiratory alkalosis

a. Decreased Bicarbonate – 2.5 mmol/lper 10mmHg PaCO2 below 40 to min 18

iv. Chronic respiratory acidosis – HCO3 – 4mmol/lper 10mmHg PaCO2 above 40 to max 36

1. Metabolic acidosis

a. Appreciation of acid base (specifically metabolic acidosis) shall be taken in the context of the anion gap

Anion Gap = (Na + K) (Cl + HCO3) ~ 12-17 mmol/l

UNMEASURED CATAIONS	UNMEASURED ANION
Mg+++ ~ 1.2 mmol/l	Albumin ~ 15 mEq/l
Ca++ ~ 2.2 mmol/l	H2PO4- ~ 2 mmol/l

IgG Small HSO4- ~ 1 mmol/l Organic ~ 5mEq/l~7.0mEq/1~23 mEq/l

b. This allows classification of metabolic acidosis into raised or normal anion gap acidosis.

NB: While this is a classical "text book" subdivision, one shall be aware that the measurement of Chlorides in the lab is highly variable and assessment of the anion gap shall always be viewed within the clinical context

c. Aetiology of raised anion gap

i. Renal failure (rarely more than 23)
ii. Lactic acidosis-types A and B * normal AG does not exclude a lactic acidosis
iii. Ketoacids (Hydroxy-butyrate, Acetoacetate): Diabetes Mellitus, starvation, alcohol
iv. Rhabdomyolysis- organic acids

v. Drugs/poisons:
Aspirin- Salicylate, Lactate, Ketones
Ethanol- Acetoacetate, Lactate
Methanol- Formate (Formaldehyde), Lactate
Methanol- Formate, Acetate, Lactate, Pyruvate
Ethylene Glycol- Oxalate
Fructose- Lactate

c. Aetiology of low or normal anion gap

i. Hyperchloraemic metabolic acidosis

a. Resolving Renal failure
b. Resolving DKA

c. Renal tubular acidosis/ Carbonic Anhydrase inhibitors
d. Mineralocorticoid deficiency
e. Pancreatic, enteric fistulae
f. Ureterosigmoidostomy
g. IV HCL, Ammonium Chloride, Arginine

ii. Metabolic alkalosis due to Bicarbonate gain

iii. Hypoalbuminaemia
iv. Myeloma – IgG has positive charge, thus decreased AG
v. Increased Magnesium or Calcium (rarely)
vi. Artifically elevated Chlorides
vii. Hyperlipidaemia (questionable)

e. Management

i. High anion gap

a. Treat the underlying cause
b. No indication for Bicarbonate

ii. Normal anion gap

a. Treat underlying cause
b. Replace Biocarbonate serum level and losses

1. Approx.deficit = (24 – (HCO3) = 4 mmol/l deficit = (24 – 4) x (70 x 0.6) = 840 mmol (=ml of standard Biocarbonate solution) replace 1/3-1/2 of this amount then remeasure blood gases

2. Metabolic alkalosis

a. Aetiology / Classification

i. Common causes:

a. Diuretics
b. Vomiting
c. Post-hypercapnia for more than 48 hours
d. Any fluid loss replaced with insufficient sodium H+ excretion (Contraction alkalosis)
d. Commonly associated with hypovolaemia and / or hypokalaemia

however, actual causation by these is debated

ii. Increased proton losses: acid loss is either renal or GIT
a. Renal
1. Raised sodium reabsorption (hypovolaemia, dehydration, etc)
2. Cushing's syndrome, exogenous steroids
3. Hyperaldosteronism Primary / Secondary
4. Batter's syndrome (JGA hyperplasia)
5. Liddle's syndrome
6. Hypercalcemia / Hypomagnesaemia NDI
7. Drugs: steroids, diuretics, Carbenoxolone

b. GIT
1. N/G suctioning, protracted vomiting
2. Diarrhoea

iii. Increased bases
a. Administration of sodium bicarbonate
b. Metabolism of exogenous acid anions – Citrate, Lactate, Acetate
c. Milk / alkali syndrome
d. Renal conservation of Bicarbonate (Acidosis, Hypercarbia)

iv. Factors tending to maintain an alkalosis
a. Hypovolaemia
b. Hypokalaemia

c. Hypochloraemia
d. Hypomagnesaemia
e. Chronic hypercapnia
f. Mild chronic renal failure

b. Management

i. Correct hypovolaemia a functional ECF volume is essential for the correction of alkalosis
ii. Inotropic support of cardiac output and GFR
iii. Correct decreased Potassium, Magnesium, Phosphate
iv. Consider Acetazolamide if the alkalosis is persistent: provided the above are corrected

3. Respiratory acidosis

a. Etiology
i. Any cause of hypoventilation leading to respiratory failure
ii. May be acute or chronic in nature

b. Restore ventilation
i. Restore ventilation
ii. No indication for Bicarbonate

4. Respiratory alkalosis

a. Etiology
i. Any cause of hyperventilation in ICU
ii. Early hypoxia
iii. Anxiety, hysteria
iv. Prescribed hyperventilation (rarely indicated)
v. Neurogenic hyperventilation

b. Management
i. Treat underlying cause
ii. Neurogenic hyperventilation is a marker of severity of head injury

I. Special Considerations

i. Severe gastrointestinal hemorrhage

a. General Management Principles

Medications for ulcer treatment and prophylaxis have reduced the incidence of stress gastritis and severe upper gastrointestinal (GI) bleeding. However, when present such bleeding can be life-threatening and requires early surgical consultation. The distinction between upper and lower GI sources is

important in determining the appropriate diagnostic/therapeutic approach. The ligament of Treitz is the anatomic landmark that separates the upper from the lower GI tract when discussing hemorrhage.
Typically, patients with life-threatening GI hemorrhage are older and have other chronic organ system disease(s). Therefore, the critical consequences of GI hemorrhage, hypotension and anemia, may be poorly tolerated and may lead to other systemic manifestations of poor oxygen delivery, such as myocardial ischemia. Prompt resuscitation and early diagnosis (even during resuscitation) are needed to prevent these secondary consequences.

Steps in rapid assessment and acute intervention include the following:

1. Verify the patient with active hematemesis has airway protective reflexes to maintain adequate ventilation and oxygenation. This will be critical if endoscopy is anticipated and sedation for the procedure will be used. Endotracheal intubation may be advisable if the patient's level of consciousness is altered, if copious of frequent emesis is occurring, or for protection of the airway during endoscopy.

2. Obtain intravenous access by means of a large-bore catheter peripherically or a large central venous line. Frequently, more than one intravenous access is needed to match the volume of resuscitation fluid to hemorrhagic losses.

Isotonic fluids (lactated Ringer's solution or normal saline) and blood products are the resuscitation fluids and choice.

4. Monitor availability of blood products on the basis of frequent assessment of visible blood loss, vital signs, hemoglobin/hematocrit, and laboratory tests for coagulation (PT, PTT, platelets, and other specialized tests (i.e., fibrinogen) as needed). A hemoglobin level of 9 to 10 g/dL (90 to 100g/L) should be maintained and coagulopathies corrected. If a reserve (e.g., 4 units PRBC) of blood products cannot be immediately available, early patient transfer should be planned.

4. Institute appropriate monitoring of systemic oxygenation (pulse oximetry), blood pressure, and, if needed, central venous (or pulmonary artery occlusion) pressure. The presence of orthostatic changes in blood pressure, tachycardia, altered level of consciousness, and decreased urine output all suggest that significant blood loss has already occurred.

5. Plan for rapid diagnostic evaluation, potential surgical intervention, or transfer of the patient.

b. Severe Upper Gastrointestinal Hemorrhage

Severe upper GI hemorrhage is diagnosed by hematemesis or the presence of blood in the gastric aspirate, although 10% to 15% of patients with duodenal ulcer may have little or no blood in the
gastric aspirate. When obtaining the patient's history and physical examination, special attention should be given to a history of previous upper GI bleeding, previous ulcer disease, alcohol consumption, stigmata of cirrhosis, coagulation disorders, and use of aspirin, nonsteroidal anti-inflammatory durgs, or anticoagulants. Common causes of upper GI hemorrhage include duodenal ulcer, gastric ulcer, esophageal, varices, Mallory-Weiss tear, and gastritis.

Endoscopy is needed to establish the diagnosis, and endoscopic therapy may control hemorrhage and reduce rebleeding. If endoscopy is available, cleansing of the stomach will likely be needed. Placement of a naso or orogastric tube, or a large tube (e.g., Ewald tube) will be needed to perform gastric lavage and remove clots. If endoscopy is not quickly available, consider transfer to a facility with endoscopic capabilities.

Intravenous vasopressin or octreotide therapy may be considered when uncontrolled variceal bleeding is suspected and endoscopy is not available. Octreotide is the agent of choice because of its favourable side effect profile. Nausea and abdominal pain are sometimes associated with bolus doses (25 to 100μg), but significant adverse effects are uncommon. Maintenance infusions of 25 to 50μg/hr continued for 48 to 72 hours may be effective in

stopping acute variceal bleeding as well as early rebleeding from varices. Vasopressin is an alternative choice, but may cause coronary artery vasospasm, angina, or hypertension. Concomitant nitroglycerin may prevent the deleterious effect of vasopression on the coronary circulation.

c. Severe Lower Gastrointestinal Hemorrhage

Frequent causes of lower GI hemorrhage are diverticular disease, carcinoma of the colon, inflammatory bowel disease, colonic polyps, and vascular ectasia. Evaluation should include a thorough history and physical examination with special attention to a history of diverticular disease, inflammatory bowel disease, previous abdominal aortic aneurysm repair (may suggest life-threatening aortic-enteric fistula), or the presence of a coagulation disorder. Physical examination must include inspection and a careful rectal examination to identify hemorrhoids orrectal carcinoma. On occasion, upper GI hemorrhage may mimic lower GI hemorrhage when associated with rapid bowel transit of blood.

Gastric aspiration or upper GI endoscopy should be performed to eliminate an upper GI source of hemorrhage. A guaiac-negative nasogastric aspirate that contains bile makes an upper GI bleeding source unlikely. Lower GI endoscopy is important for diagnosis, therapy, anticipating rebleeding, and planning other diagnostic interventions or surgery. If endoscopy is unavailable, transfer of the patient or direct surgical intervention will be dictated by the patient's condition. Angiography with embolization therapy may be considered for unstable patients or those who are poor surgical risks. Causes of lower GI bleeding in pediatric patients are shown in Table.

For Pediatric Patients

Causes of lower gastrointestinal bleeding in pediatric patients

1 month to 1 year	Meckle's diverticulum
1 to 2 years	Intussusception
2 to 12 years	Polyps, child abuse, inflammatory bowel disease

d. Continuing Care

If the hemorrhage is controlled initially, continuing vigilance is required to detect rebleeding. Careful monitoring of physiologic variables, laboratory parameters, and external signs of blood loss are required in an environment where adequate, trained staff and blood bank resources are immediately available.

ii. Pulmonary Embolish

a. Diagnosis

The patient's history and clinical findings are unreliable for diagnosis of a pulmonary embolus (PE). Risk factors for pulmonary embolism are shown in table and often contribute to a high index of suspicion for PE.

Risk factors for pulmonary embolism

Heart failure	Surgery
Advanced age	Immobility, paralysis
Trauma	Previous pulmonary embolism or deep
Obesity	venous thrombosis
Estrogens, pregnancy	Hypercoagulable disorders
Malignancy	

For Pediatric Patients

PE does occur in critically ill children, but is diagnosed much less commonly than in adults. PE may go unrecognized in critically ill patients. PE is not commonly recognized in obese adolescents immobilized for long periods.

The classically described combination of dyspnea, plueritic chest pain, and hemoptysis occurs in a minority of patients with PE. Routine blood studies are nondiagnostic. Chest radiographs frequently are unremarkable but may show nonspecific findings of atelectasis, pleural effusion, and infiltrates. The ECG usually shows nonspecific ST-T wave changes, and a pattern of acute cor pulmonale is present infrequently. Sinus tachycardia and premature atrial contractions are the most frequently encountered arrhythmias. The most significant information provided by the ECG is often the eclusion of other potential sources of chest pain, such as acute ischemia or pericarditis. Hypoxemia, a nonspecific finding in cardiopulmonary disease, is commonly present, but a normal PaO2 value or normal alveolar-arterial oxygen tension

difference does not rule out PE. Signs and symptoms of PE are shown in table.

Clinical manifestations of pulmonary embolish

	Cough
Dyspnea	
Tachypnea	Hemoptysis
Tachycardia	Fever (usually low-grade)
Chest pain	

A correct diagnosis of PE is essential because appropriate therapy decreases mortality. The present diagnostic strategy, based upon the large. Prospective Investigation of Pulmonary

Diagnosis (PIOPED) study encourages the physician to formulate a low, uncertain, or high clinical suspicion of embolus. A radionuclide perfusion lung scan is typically combined with a chest radiograph (in patients with underlying cardiopulmonary disease) or a ventilation scan and interpreted by a radiologist who is familiar with the standardized criteria for negative, low-probability, intermediate, and high-probability scans. The recommended algorithm is then utilized to propose additional diagnostic tests or therapy. A positive lower extremity study (e.g., compression ultrasound, contrast venogram, impedance plethysmography, or radionuclide venogram) indicates the need for treatment, since the treatment for deep venous thrombosis is the same as that for PE. Other diagnostic methods for PE include echocardiograph or high-speed helical (spiral) CT scans of the pulmonary circulation. A negatie D-dimer result may be helpful in patients with a low clinical suspicion for PE.

b. Therapy

Treatment usually can be limited to anticoagulation. Low-molecular-weight heparins (dalteparin, enoxaparin, nadroparin, tinzaparin) can also be used to effectively treat PE and offer the benefit of convenient dosing. In patients with suspected PE and no contraindications to anticoagulation, baseline activated partial thromboplastin time (APTT), prothrombin time (PT), and complete blood count should be obtained and heparin initiated while diagnostic tests are being obtained. Contraindications to heparin therapy include recent major trauma with hemorrhage, recent central nervous system (CNS) hemorrhage or infarction, active GI bleed, and clinically significant heparin-induced thrombocytopenia. The APPT should be monitored when using unfractionated heparin to achieve a value 1.5 to 2.0 times the mean normal value. Heparin therapy can be discontinued after 5 to 6 days if the INR with warfarin has been therapeutic for 2 days. In massive PE, a longer period of heparin therapy (~ 10 days) is often recommended. Oral warfarin therapy is started on day 1 and adjusted to achieve and INR 2.0 to 3.0 oral anticoagulation should continue for at least 3 months.

A massive pulmonary embolus can be treated with thrombolysis if the patient is in shock, is unresponsive to other supportive care, and does not have an absolute contraindication to such therapy. The use of thrombolytic agents in the treatment of PE should be highly individualized, and clinicians should have some latitude in using these agents. In general, patients withHemodynamic instability who are at low risk to bleed are the best candidates for thrombolytic therapy. Tissue plasminogen activator (t-PA) is preferred at a dose of 100mg infused over a 2-hour period, but none of the thrombolytics has been shown to improve mortality in this extreme situation. Surgical embolectomy or extraction or fragmentation of the embolus by transvenous catheters requires specialized expertise not commonly available, and associated mortality is high.

An inferior vena cava filter should be considered to prevent further emboli when

- there is a strong contraindication to anticoagulation
- emboli recur during anticoagulation
- bleeding occurs during anticoagulation

c. Clinical Issues

for the patient with manifestations consistent with hypoxemia (e.g., Tachycardia, hypotension, anxiety, agitation), always consider performing an arterial blood gas measurement to validate the oxyhemoglobin saturation measurement.

iii. Aortic dissection

a. Clinical Presentation

Aortic dissection typically occurs after trauma or in hypertensive patients in their fifth and sixth decades of life, but can occur in your adults with

Marfan's or Ehlers-Danlos syndrome. Symptoms are often severe and usually include unrelenting chest pain, frequently associated with back or epigastric pain. A patient with moderate to severe hypertension, severe chest pain, and an enlarged mediastinum on chest radiograph has an aortic dissection until proven otherwise. Involvement of major branches of the aorta can lead to coma, hemiplegia, extremity ischemia, spinal cord infarction, or paraplegia. Aortic dissection is frequently misdiagnosed as acute myocardial infarction, or paraplegia. Aortic dissection is frequently misdiagnosed as acute myocardial infarction, PE, stroke, esophagitis, pancreatitis, peptic ulcer disease, biliary colic, and ureteral colic.

New-onset limb ischemia or aortic valve insufficiency may also suggest aortic dissection. Physical examination should therefore include careful

auscultation for new murmur of aortic insufficiency, assessment for asymmetric blood pressure or pulses in upper extremities, and
careful palpation of pulses in all extremities for significant asymmetry. The diagnostic standard is angiography, if available. Computed tomography scanning with rapid-sequence contrast and tranesophageal echocardiography may also be used for diagnosis.

b. Management

Control of blood pressure and heart rate is critical. As immediate surgical consultation is obtained or plans are initiated for transfer, treatment with parenteral antihypertensive agents should be initiated. The combination of intravenous propranolol and sodium nitropresside or intravenous labetalol is the treatment of choice. Intravenous esmolol is an alternative to propranolol. If sodium nitroprusside is necessary to lower blood pressure, concomitant β-blockade is required to reduce the rate of rise (dp/dt), or shear force, of the blood pressure, and intra-arterial pressure monitoring is usually necessary. The clinical goals are to relieve pain and to lower the systolic pressure to 100 to 120 mmHg, but to maintain a mean arterial pressure of 60 to 70 mmHg. Medical management is always a temporizing measure until complete diagnostic studies and surgical evaluation have been finalized.

iv. Temperature-related illness

a. Heat Stroke

Heat stroke is the most common cause of life-threatening hyperthermia. In addition to environmental heat and humidity, predisposing factors include cardiovascular disease, strenuous exertion, obesity, skin or sweat gland abnormalities, and drugs (e.g., alcohol, anticholingergics, sympathomimetics, β-adrenergic blockers) that limit the patient's ability to dissipate body heat. The patient's core temperature is usually > 40°C (>140°F). Additional clinical manifestations in heat stroke include major CNS dysfunction (e.g., confusion,

seizures, comma), tachypnea, and tachycardia. Hypotension is common, and sweating may be absent or present.

Treatment of heat stroke is directed toward replacement of intravascular volume, immediate reduction of the core body temperature, and reversal or prevention of complications. Resuscitation measures should be instituted as outline below.

- Protect the airway
- Supply oxygen
- Administer intravenous fluids; the type of amount depend on assessment of electrolytes, volume status, and vital signs. Hypo- or hyperkalemia, hypernatremia, hypocalemia, and hypophosphatemia may occur.
- Cool the patient immediately (monitor with a rectal or esophageal thermistor capable of recording high temperatures).

Evaporative cooling by misting the unclothed patient with tepid (not cold) water and circulating air with a fan is easy to institute in most environments. An ice water bath is another option for cooling, but this method may induce shivering and vasoconstriction, thus generating more body heat and slowing its dissipation. Alternatively, application of ice packs or ice water soaks to the maxillae and groin can be used. Antipyretics are ineffective.

b. Hypothermia

Hypothermia is usually due to exposure or immersion and is defined as < 35° C (<95°F) core temperature. Predisposing factors include extremes of age, trauma, erythrodermas, and drugs such as alcohol, barbiturates, phenothiazines, and benzodiazepines. Clinical manifestations vary with the decrease in core temperature. Neurologic changes range from impaired judgement to coma. Muscle tone is increased, and shivering may be present at core temperatures from 30° to 32° (86° to 89.6°F). Cardiorespiratory

findings include arrhythmias (including ventricular fibrillation), hypotension, and hypoventilation. Osborn or J waves may be seen on the ECG. Resuscitation measures should be instituted as follows and should be continued until rewarming occurs.

- Protect the airway
- Institute cardiopulmonary resuscitation if there is no evidence of perfection
- Remove wet clothes and apply blankets
- Avoid excessive patient manipulation or stimulation that may precipitate arrhythmias. Special care is needed when inserting central venous catheters in the chest so that the wire or catheter does not enter the heart. Pulmonary artery catheters are relatively contraindicated.
- Administer warm, intravenous fluids (D5 normal saline). The patient is often hypovolemic because of a cold diuresis.
- Insert rectal or esophageal temperature probe capable of recording low temperatures.
- Initiate rewarming

The choice of a rewarming technique depends on the patient's temperature and response to simple measures. Passive external rewarming blankets or insulting materials, as well as active external rewarming with heating blankets, is useful in mild hypothermia (32° (<89.6°F) and/or life-threatening hemodynamic instability. Active, external rewarming with warm water

immersion, heat lamps, convective heaters to increase room temperature, or forced air blankets or warming devices may be considered in moderate hypothermia (30° to 32° (86° to 89.6°F)) when advanced facilities are unavailable. Active core rewarming is preferred in severe hypothermia (<28° C (82.4°F), loss of cardiac activity, or failure to elevate temperature by 1° to 2° C (1.8° to 3.6°F) per hour with other methods. Intubation and inhalation of heated, humidified oxygen is effective and is readily available. Irrigation of the stomach and / or bladder with warm isotonic fluids is only marginally effective and may distract from more effective therapy. Consultation should

be considered to institute peritoneal lavage, pleural lavage, warming via a hemodialysis machine or cardiopulmonary bypass in the most unstable patients. The intramuscular and subcutaneous routes of medication administration should be avoided, as absorption is erratic.

With extreme hypothermia, the blood pressure and pulse may be very difficulty to record. ECG monitoring and careful attention for other signs of life (e.g., movement, respirations) may demonstrate a stable physiology in such a patient with a profound reduction in oxygen consumption. Vasoactive drugs should be used cautiously because of their arrhythmogenic potential. If cardiopulmonary resuscitation is required, recall:

- Defibrillation may be ineffective until core temperature is above 28° to 30° C (82.4° to 86°F)
- Adrenergic drugs, likewise, may be ineffective below 30° C (86°F)
- Full resuscitative efforts should generally be continued until the core temperature is > 30° to 32°C (86° to 90°F)

- The decision to terminate resuscitation is individualized on the basis of the circumstances causing the hypothermia and other factors

J. Critical care in pregnancy

i. Introduction

The pregnant women can present for critical care support in two major ways. First, some disease states are unique to pregnancy, such as eclampsia and the hemolysis-elevated liver enzymes-low platelet count (HELLP) syndrome, and require significant immediate therapy. Second, other critical illness not unique to pregnancy, such as thromboembolic disease, cardiac disease, and

trauma, can be precipitated or aggravated by pregnancy and may have different manifestations during pregnancy or required alterations in planned treatment.

ii. Physiologic alterations (core)

a. Cardiovascular

Blood volume increases in each trimester during pregnancy. Cardiac output increase 30% to 50% during normal pregnancy, with most of the increase occurring during the first trimester. Cardiac output increases as a result of increased stroke volume during the first and second trimesters and by an increase in heart rate during the third trimester. A significant decrease (25% to 30%) in cardiac output may occur in the third trimester if the patient is placed in the supine position, owing to compression of the aorta and inferior vena cava by the gravid uterus, increasing afterload and restricting venous return to the heart. Filling pressures (central venous and pulmonary artery pressures) typically do not change during pregnancy. Despite increase in cardiac output, blood pressure (BP) decreases during a normal pregnancy (BP is lowest during the second trimester) typically to not change during pregnancy. Despite increases in cardiac output, blood pressure (BP) decreases during a normal pregnancy (BP is lowest during the second trimester) as a result of diminished systemic vascular resistance (SVR) secondary to the vasodilating effects of progesterone. Peak reduction in BP occurs at 24 weeks; systolic pressures are reduced by 5 to 10 mmHg and

diastolic pressures by 10 to 15 mmHg. It is abnormal for blood pressure during pregnancy to exceed nonpregnant values.

b. Pulmonary

Pulmonary changes in pregnancy include an increase in tidal volume of approximately 40% and a decrease in functional residual capacity (FRC) by 25%. The reduction in FRC predisposes the patient to atelectasis if critical

illness develops. The combination of decreased FRC and increased oxygen consumption during pregnancy diminishes the oxygen reserves of the mother and subsequently increases the hypoxic risk to the fetus in response to maternal hypoventilation or apnea. Oxygen requirements increase by approximately 30 to 40 mL/min in pregnancy and are met by an increase in minute ventilation, primarily as a result of increased tidal volume. The increase in minute ventilation results in a mild compensated respiratory alkalosis with a decline in the PaCO2 to ~30 torr (4.0 kPa). The pH does not change secondary to renal compensation that results in a decrease in the serum bicarbonate concentration.

c. Gastrointestinal

Hormonal and anatomic changes in pregnancy affect the gastrointestinal tract. A reduction in lower esophageal sphincter pressure and a resulting increase in gastroesophageal reflux contribute to an increased risk for aspiration, starting at the end of the first trimester. Alterations in gastric motor function may cause nausea, vomiting, and dyspepsia.

d. Hematologic

The 40% to 60% increase in plasma volume that occurs by the third trimester is associated with an increase in red cell mass of only 25% at term. The disproportionate risk in plasma volume results in diluational anemia; hemoglobin concentration is ~ 11g/dL (110g/L) at 24 weeks. The white blood cell count climbs to 10,000 cells/min^3 at term, with a slight reduction in

platelet count. Concentrations of all clotting factors except XI, XIII, and antithrombin III increase in pregnancy. Fibrinogen levels may be as high as 600 mg/dL (6.0g/L) at term; levels < 150 mg/dL (<1.5g/L) are considered abnormal. Although coagulation tests and bleeding times do not change, these compositional changes result in a hypercoagulable state that, in

association with venous stasis and vein wall trauma, elevates the risk for thromboembolism.

iii. Hypertensive disorder (Core)

a. Diagnosis

1. Preeclampsia (Pregnancy-Induced Hypertension)

The diagnosis or preeclampsia is defined by the development of hypertension with proteinuria, with or without edema, after 20 weeks of gestation.

Preeclampsia is classified as severe if at least one of the following signs is present:

Resting BP $\geq$ 160 mmHg systolic or 110 mmHg diastolic at any time, or 140 mmHg systolic or 90 mmHg diastolic associated with any of the complications listed below.

- Proteinuria $\geq$ 5g/24hr or 3+/4+ on urine dipstick
- Oliguria < 30 cc/hr for 3 consecutive hours
- Systemic symptoms, including pulmonary edema, right upper quadrant pain, impaired liver function, headache, visual changes, or thrombocytopaenia.

2. Eclampsia

Eclampsia is defined as preeclampsia with seizures. In some cases, pregnancy-induced hypertension may present initially as eclampsia.

Although seizures are the most dramatic manifestation of preeclampsia, other intracranial catastrophes such as hemorrhage or stroke are more likely to cause death. Eclampsia usually occurs after 20 weeks of gestation or within 48 hours after delivery but should be considered in the differential

diagnosis of seizures up to 14 days after delivery. Eclampsia occurring > 48 hours postpartum is more likely to be misdiagnosed.

b. Management

1. General Guidelines

Patients with eclampsia or severe preeclampsia require hospital admission. Administration of magnesium sulfate, control of blood pressure, and maternal/ fetal monitoring should be initiated. Consultation with an obstetrician should be sought. Issues such as ICU admission, management, and delivery of the fetus should be discussed with an obstetrician or critical care physician as soon as possible. Preventing maternal injury, ensuring maternal and fetal oxygenation, and initiating seizure prophylaxis are important aspects of therapy. The therapy of choice is delivery, but the maturity of the fetus must be considered. In most cases of severe preeclampsia occurring after 32 weeks of gestation, the baby should be delivered. Consultation with a maternal-fetal medicine specialist is recommended.

2. Seizure Prophylaxis

Magnesium sulfate (20% solution) is used in preeclampsia in an attempt to avoid progression to eclampsia. This drug does not jeopardize the fetus and can be given intramuscularly or intravenously. Magnesium sulfate therapy is usually initiated when the diagnostic BP is > 100
mmHg and signs of impending seizure, such as visual blurring, scotomata, or hyperreflexia, or signs of severe preeclampsia are present. Although magnesium sulfate is used prophylactically in the U.S. in patients with preeclampsia, other physicians may use magnesium sulfate only for

treatment of eclamptic seizures. Clinical studies are under way to better define the appropriate use of magnesium sulfate.

Administration of magnesium sulfate by the intravenous route is preferred because serum levels are more stable and there is less material discomfort. A loading dose of 4 to 6 g in 200 to 250 cc normal saline over 10 to 15 minutes is followed by an intravenous infusion of 1 to 2 g/hr. Magnesium levels are checked 2 to 4 hours late and should be 2.0 to 3.5 mmol/L (4 to 7 mEq/L). Maternal respiratory rate, deep tendon reflexes, level of consciousness, and urine output are monitored regularly. Respiratory depression, somnolence, or loss of patellar reflexes suggest magnesium levels is excess of the therapeutic range (>3.5 mmol/L or 7 mEq/L). Since magnesium is excreted renally, the infusion rate should be decreased if urine output drops. The maintenance infusion should be decreased or withheld on the basis of the serum creatinine level. Normal pregnant women have a creatinine level of 0.5 to 0.9 ml/dL (40 to 80µmol/L). The antidote for magnesium toxicity is 1 gm calcium chloride (10 mL of 10% solution) given intravenously over severe minutes.

3. Blood Pressure Control

The goal of therapy is to lower the systemic BP to a point at which maternal status is stabilized. Although it is not necessary to lower BP to normal levels, diastolic BP should be reduced to 90 to 100 mmHg. In patients with extremely elevated BP, the mean arterial pressure should be lowered gradually in increments of 10% to 15% at one time. Antihypertensive therapy that results in precipitous drops in BP could further comprise an already stressed fetus by shunting blood away from the placental circulation. Drugs that are generally used are hydralazine (2.5 to 5 mg administrated as a slow intravenous push every 15 to 20 minutes for three doses) and labetalol (20 mg intravenously initially, and titrated every 10 to 15 minutes). If the initial 20-mg dose of labetalol is not effective, 40 mg should be given. If the 40-mg dose does not lower the BP to the desired level, it should be followed by an 80-mg dose. Diuretics should usually be avoided because most preeclamptic patients have constricted plasma volume. Calcium-channel blockers have

been used extensively in preeclampsia; nifedipine (10 to 20 mm orally every 4 to 6 hours) is the most commonly employed agent. Use of calcium-channel

blockers, especially nifedipine, is being reevaluated because of reported myocardial ischemia and sudden death in the acute treatment of hypertension. The angiotensin-converting enzyme (ACE) inhibitors are contraindicated in pregnancy because of associated fetal and neonatal complications. Nitroprusside has also been used, despite reports of cyanide toxicity in animal models. Nitroglycerin is less effective in preeclampsia because of its primary venodilator effect.

4. Supportive Measures

Cardiogenic and noncardiogenic pulmonary edema can occur during severe preeclampsia. Treatment includes supplemental oxygen to maintain maternal PaO2 <70(>9.3kPa) with oxygen saturations (SpO_2) $\geq$ 94% to prevent fetal hypoxia and progressive acidosis. If necessary, tracheal intubation and mechanical ventilation are instituted. Intubation should be approached cautiously in the pregnant women because of the increased risk of aspiration, reduced FRC, pharyngeal edema, and increased hypoxic risk to the fetus. Usually, smaller Endotracheal tubes (6.0 or 6.5mm) are necessary. With cardiogenic pulmonary edema, fluid restriction and diuretics are often the initial primary treatment. Since preeclamptic and eclamptic patients are frequently depleted intravascularly, invasive hemodynamic monitoring may be required to optimize management. This is an especially important measure in patients who have altered pulmonary capillary permeability.

Oliguria occurs frequently in severe preeclampsia and eclampsia. Intravenous fluid challenges should be instituted cautiously. The empiric use of diuretics in the absence of invasive hemodynamic monitoring to assess intravascular volume is discouraged. Patients who do not respond to fluid challenges need further assessment. Noninvasive techniques such as echocardiography can be used to assess the cardiac output, central venous pressure, and ejection fraction. Repeated fluid boluses will usually be tolerated by preeclamptic patients with a good ejection fraction and cardiac output. Most preeclamptic

women with oliguria will respond to 1 to 2 liters of crystalloid without the need of invasive monitoring. Failure of the patient to respond to repeated fluid challenges, or the presence of cardiac failure, should prompt consideration of intravascular hemodynamic monitoring. Vasodilator therapy may be beneficial if intravascular volume is adequate.

5. Monitoring

For all patients, BP level should be monitored regularly. When magnesium sulfate is used, monitoring consists of checking reflexes, respiratory rate, and periodic magnesium levels. Invasive hemodynamic monitoring of preeclamptic patients is infrequently required. Central venous pressures measurements are not reliable in this patient population.

iv. HELLP syndrome (Core)

The HELLP syndrome identifies a severe, life-threatening condition in pregnancy. The mnemonic "HELLP" stands for

- Hemolysis – hemolytic microangiopathic anemia with an abnormal peripheral smear, a total bilirubin > 1.2 mg/dL (21 μmol/L), or serum lactate dehydrogenase (LDH) level > 600 U/L;
- Elevated Liver enzymes – Aspartate aminotransferase (AST)>70U/L, or LDH>600U/L; and
- Low Platelet count - < 150,000/mm^3

Variations of the syndrome do not necessarily include all manifestations. The HELLP syndrome can present with a variety of nonspecific clinical signs and symptoms, including epigastric or right upper quadrant pain, gum or nose bleeds, petechiae, malaise, nausea, and vomiting. Most patients with HELLP syndrome present at a gestational age of 27 to 36 weeks. Postpartum presentations also occur, usually within 1 to 2 days after delivery.

Preeclampsia often precedes the HELLP syndrome, but one third of patients with HELLP syndrome have no hypertension.

The HELLP syndrome is part of the fibrinolysis or hemolysis portion of the preeclamptic syndrome. Thrombocytopaenia, DIC, intracerebral hemorrhage, liver rupture, renal failure, and HELLP are the different manifestations. HELLP syndrome can sometimes be confused with an acute fatty liver of pregnancy, thrombotic thrombocytopenic purpura, and the adult hemolytic uremic syndrome. Laboratory tests are helpful in differentiating these conditions are listed in Table. HELLP syndrome is almost always an indication for urgent delivery because of increased fetal and maternal morbidity and mortality. If possible, management of the HELLP syndrome should occur in a tertiary care facility. Treatment Including supportive care, intravenous magnesium sulfate, and antihypertensive therapy for diastolic BP > 10 mmHg. Plasmapheresis has also been performed when symptomatic thrombocytopaenia continues after aggressive platelet replacement, but this option is rarely used. Patients complaining of persistent, severe, or worsening, epigastric or right upper quadrant pain should be carefully examined for spontaneous rupture of the liver. Computed tomography or MRI can be useful to diagnose intrahepatic bleeding.

Laboratory differentiation of acute fatty liver and HELLP syndrome

Test	**Acute Fatty liver**	**HELLP**
Fibrinogen	↓	Normal or↑
Glucose	↓	Normal
Ammonia	↑	Normal

v. Peripartum cardiomyopathy (Supplemental)

a. Clinical Manifestations

Peripartum Cardiomyopathy is defined as congestive heart failure occurring during the last month of pregnancy or in the first 5 months postpartum. Clinical indicators include severe progressive dyspnea, progressive orthopnea, paroxysmal nocturnal dyspnea, or syncope with exertion. Signs include general or chamber-specific cardiomegaly seen on a chest radiograph, evidence of pulmonary hypertension, murmur, prominent jugular vein distension, cyanosis, clubbing, or arrhythmia. Heart failure without identifiable cause in a patient between the last month of pregnancy and 5 months postpartum suggests peripartum Cardiomyopathy. The course tends to be more severe in older patients of higher parity with later onset of symptoms after delivery.

b. Management

Initial evaluation of the patient with possible peripartum Cardiomyopathy includes a chest radiograph, electrocardiogram (ECG), and echocardiogram. Therapy includes bed rest, sodium restriction, diuretics, and possibly vasodilators. Patients who present with fulminant pulmonary edema and cardiac decompensation often require invasive hemodynamic monitoring for careful fluid management, intravenous inotropic support, and afterload reduction. Useful drugs include digoxin as an inotropic agent and ACE inhibitors for afterload reduction (ACE inhibitors are contraindicated prior to delivery). If symptoms develop in the antepartum period, consultation between the obstetrician, critical care physician, and anesthesiologist can guide decisions regarding early delivery. Anticoagulation should be considered in the pregnant woman with peripartum Cardiomyopathy, enlarged cardiac chambers, and an ejection fraction < 35%, since the

increase in both systemic and pulmonary emboli is significant. Postpartum cardiac function

returns to normal in approximately 50% of patients. The 50% of patients who continue to have symptoms have a mean survival time of ≤ 5 years.

vi. Thromboembolic disease

Thromboembolic disease is a significant factor is material deaths. The incidence of thromboembolic disease in pregnancy and the immediate postpartum period is five times the incidence in the nonpregnant woman. A higher risk occurs with increased parity, cesarean section, operative vaginal delivery, previous deep venous thrombosis, and increased age.

Although manifestations of pulmonary embolism in pregnant women are similar to those in nonpregnant women, the physiologic changes of pregnancy complicate evaluation. Lower-extremity edema, leg pain, and dyspnea are common findings in pregnancy and create a diagnostic dilemma for the clinician. If a chest radiograph is obtained to rule out other pulmonary problems, such as pneumonia, the fetus must be shielded. Doppler scanning of the lower extremities can be unreliable because of venous stasis and pooling. Ventilation/ perfusion (V/Q) scanning is reliable in a pregnant woman, but angiography is often necessary when (V/Q) scanning results are indeterminate. A Spiral CT Angiogram can be useful, especially with centrally located emboli. Heparin therapy should be initiated when the diagnosis of pulmonary embolism is suspected and should be continued if the diagnosis is confirmed.

The treatment of pulmonary embolism in the pregnant patient parallels that in thenonpregnant patient, except that coumadin is relatively contraindicated in pregnancy and absolutely contraindicated during the first trimester, when the risk of teratogenicity is greatest. Instead, unfractionated heparin can be administered intravenously by weight-adjusted dose regimen to achieve an activated partial thromboplastin time (aPTT) of 1.5 to 2.5 times control. After

at least 5 days of intravenous heparin, treatment can be converted to subcutaneous administration, starting at 5000 IU of unfractionated heparin every 12 hours and aiming for the same aPTT goal measured 6 hours after

administration. Indwelling Teflon catheter (peripherally inserted central catheters) can be used during pregnancy to facilitate heparin administration. Low molecular weight heparins (LMWH) are safe for the fetus and can be used for the treatment of acute Xa levels must be monitored weekly. Patients receiving heparin are at risk for heparin-induced thrombocytopaenia (the risk is lower with LMWH) and osteoporosis. After delivery, warfarin can be substituted, for 3 to 6 months of total therapy.

Intrapartum management requires that heparin be discontinued at least 4 to 6 hours before delivery and be resumed 6 to 24 hours after delivery. The risks with intrapartum use of heparin include a significantly increased likelihood of hemorrhage with cesarean delivery, and bleeding and hematomas if a regional anesthetic is used or an episiotomy or operative vaginal delivery is performed.

vii. Severe Asthma

Asthma is the most common pulmonary condition in pregnancy. Among asthmatic patients, 50% have no change in symptoms, 30% improve, and 20% worsen. Treatment of asthma usually does not require modification during pregnancy. Supplemental oxygen should be administered if oxygenation is in question or a low pulse oximeters measurements is obtained. In severe asthmatic attacks, management with inhaled β-agonists and systemic steroids is preferred. Parenteral corticosteroids can be safely administered.

viii. Septic pelvic thrombophlebitis

Septic pelvic thrombophlebitis, characterized by infected clot(s) in the pelvic veins, occurs in the peripartum period. Physical findings are nonspecific.

Failure of fever to respond to empiric antibiotics in a postpartum patient should prompt consideration of this condition. Evidence of systemic septic emboli (e.g., sepsis, metastatic abscesses, and septic pulmonary emboli) may be present. Ultrasonography or computed tomography (CT) studies are not

diagnostic but can occasionally show evidence of a clot. Patients are typically treated on the basis of clinical suspicion as a diagnosis of exclusion. Heparin, as outlined above, is used in addition to antibiotics for management. A reduction in fever usually occurs within 24 hours. Anticoagulation therapy is continued for 3 to 6 months postpartum.

ix. Trauma in pregnancy

Treatment priorities for the pregnant patient with traumatic injury are the same as those for nonpregnant patients. These are, however, unique changes that should be taken into account during clinical assessment. The gravid uterus complicates the initial abdominal assessment of the pregnant patient. The height of the uterus is roughly at the symphysis pubis at 12 weeks and the umbilicus at 20 weeks; then the height increase by 1 cm/wk thereafter up to 36 to 40 weeks, when the uterus encompasses almost the entire abdomen. Late in pregnancy, a widened symphysis pubis and widened sacroiliac joints are possible. All pregnant patients with major traumatic injuries should be admitted to a facility with surgical obstetric capabilities. When evaluating mental status, be aware that neurologic symptoms of eclampsia may mimic head injury. Venocaval compression can contribute to hypotension by restricting the return of blood to the heart. Whenever possible, the patient is placed in the left lateral decubitus position, or, at a mimimum, the right hip is raised by 4 to 8 cm to displace the uterus off the inferior vena cava. If any question of spinal injury exists, spinal alignment is maintained and the patient is “log-rolled”.

The pregnant patient can lose up to 35% of blood volume before significant tachycardia, hypotension, and other signs of hypovolaemia are seen. Therefore, the fetus may actually be in a state of hypoperfusion while the

mother's condition seems stable. An assessment of the fetal heart rate is an essential part of the initial survey. This assessment can be accomplished easily with a fetoscope or a Doppler fetoscope. A conventional stethoscope can be used to auscultate the fetal heart rate in the third trimester. If available, ultrasonography is very effective for documenting fetal cardiac

activity and function. Normal fetal hear rate are 120 to 150 beats/min, late of persistent decelerations of the fetal heart rate are an aminous sign. If ther fetus cannot be examined adequately at the facility, the patient should be stabilized and transported as soon as possible. A minimum of 4 hours of fetal monitoring is necessary after trauma.

Secondary assessment should evaluate uterine irritability (spasms of the uterus), fetal heart rate, and fetal movement, and a pelvic examination should be performed, if necessary. If there is any question of blood from the vagina, a qualified, experienced person should do a speculum examination or, preferably, contraindicated if placenta previa is a possibility.

Definitive care of the pregnant trauma patient includes adequate hemodynamic and respiratory resuscitation, stabilization of the mother, continued fetal monitoring, and radiographic studies as necessary, in addition to obstetric, critical care, and surgical consultation. If he other Rh-negative, Rh_o(D) immune globulin (Rhogam ™) should be given within 72 hours of injury, even when trauma is minimal. An assessment of the amount of fetal red blood cells in the maternal circulation by means of a Kleihauer Betke stain is advised. Obstetrical consultation for appropriate dosings if Rho (D) immune globulin is recommended.

9. MICROBOLOGY PROTOCOLS

1. Policy
    a. The diagnosis of infection in critically ill patients is both important

and difficult

b. Sepsis is the most common cause of death in critically ill patients. It shall be aggressively sought and promptly treated with surgical drainage (where indicated) and appropriate antibiotics

c. Simple preventive measures are the most important factors in the containment of hospital associated infections and development of bacterial resistance

i. Compulsory hand washing or alcohol hand rub by all staff before and after contact with a patient.

ii. Attention to aseptic technique for invasive procedures

iii. Attention to invasive procedure protocols outlined in the procedures section

iv. Avoidance of over-prescription of antibiotics and rational usage

d. Investigation shall be only be ordered on specific indications and when necessary

e. Please refer to antimicrobial guidelines for antimicrobial usage especially prophylactic and empirical usage and for combination therapy

f. De escalate antimicrobials, as soon as possible

2. Definitions

a. Systemic Inflammatory Response Syndrome (SIRS)

i. Describes the inflammatory process that occurs in response to a variety of clinical insults resulting in a clinical picture suggestive of "sepsis"

ii. The syndrome includes at least 2 of the following:

a. Temperature more than 38° C (Degrees C) or less than 36 Degrees C

b. Heart rate more than 90 beats per minute

c. Respiratory rate more than 20 breathes per minutes, or PaCO2 less than 32 mmHg

e. White cell count more than 12,000/cubic mm, or less than 4,000/cubic mm, or more than 10% immature (banded) Neutrophills

iii. SIRS is non-specific and may be due to non-infectious causes:

a. Trauma
b. Haemorrhagic shock
c. Post-operatively after major surgery
d. Pancreatitis
e. Burns

f. Blood transfusion
g. Drug reactions
h. Intracranial pathology especially intraventricular blood and Thalamic Pathology

b. Sepsis: the presence of SIRS secondary to infection
c. Septic shock: decreased vital organ perfusion/function secondary to sepsis
d. Nosocomial infection is defined as infection that occurs during hospitalization that was neither present, nor incubating on admission
e. Colonization is defined as the presence of microorganisms that do not elicit an inflammatory response

3. Microbiological Investigation

a. Routine, performed only on the clinical suspicion of sepsis

i. New pyrexia
ii. Raised TLC or marked decrease in TLC
iii. Thrombocytopenia
iv. Deteriorating gaseous exchange or pH
v. Cardiovascular instability
a. Hypotension / relative hypovolaemia
b. Increased or new inotrope requirement
vi. Oliguria or increased Creatitine

a. All samples shall be collected in sterile containers
b. Never send samples for microbiology investigations in formalin
c. If delay in delivery of sample for culture and sensitivity is Unavoidable, collect
d. or inoculate the sample in transport media
e. Wherever possible collect the sample before the start of antibiotics
f. The following information is necessary for microbiological investigations mention antimicrobial therapy given, presently or in recent past

g. Mention provisional diagnosis
h. The label shall indicate the identification of the specimen e.g. Name Identification (UHID No.), Date and Time of collection and nature of specimen
i. Containers for the samples shall be STERILE and shall not contain anything other than the specimen asked for unless advised by the Microbiology services
j. All samples shall be collected aseptically
k. Please not the Microbiology lab is accepts specimens round the clock

b. Screening

i. Urine (routine / microscopy and culture)
ii. Tracheal aspirate (gram stain and culture)
iii. Blood culture x sets
iv. Gram stain and culture of appropriate drainage fluid, for example, wound aspirate, pleural fluid etc.,

c. Other investigations, as appropriate to clinical conditions
i. Fungal cultures
ii. CSF culture and routine / microscopy
iii. Pleural tap for routine / microscopy and culture – aerobic, AFB, Fungal as appropriate, ADA
iv. Sinus X-rays

v. Bronchoscopy specimens (BAL): gram's staining, fungal smear/ KOH mount/Z.N. staining / Modified Z.N. staining, Silver Methanmine (PCP), aerobic culture, AFB culture, Fungal culture, Anaerobic, as Appropriate

d. Interpretation of results

i. Urine

a. UTI in a catheterized patients is defined as:

1. More than 10^5 bacteria/ml on positive culture of organisms, plus
2. More than 500 WBC/HPF

b. Bacteria and white cells are a normal finding in the urine in a catheterized patient

c. Treatment with antibiotics shall not result in clearance of colonization and is only indicated if it is thought that the patient is systemically unwell from this source

e. Blood cultures

i. May be contaminated by skin organisms: care shall be taken with technique:

a. Clean the skin with an alcohol or betadine swab
b. Clean the top of culture medium bottle with an alcohol swab

COLLECTION PROCEDURE

- Collection is by venipuncture or peripheral veins only
- Observe universal precautions. Wear gloves before procedure
- Blood is NOT to be taken from indwelling vascular catheter/ cannula, until and unless so specified
- Cleanse and disinfect top of appropriate culture bottles with alcohol swabs after flipping off the cap
- Apply tourniqent, select vein, and palpate site, release tourniquet

- Cleanse and disinfect site with iodine / povidone – iodine (ask for any previous history of sensitivity to Iodine / Betadine) if not possible than with 70% isopropyl alcohol and leave for 30 seconds (secs)
- With circular movement, staring from center to periphery, swab an area of diameter 5-6 cm. (I using both iodine and alcohol allow iodine to dry (60 sec) then scrub with 70% isopropyl alcohol and leave it for 30 secs)
- Apply tourniquet
- Withdraw blood from previously selected vein site
- Do not repalpate venipuncture site
- Change needle if second venipuncture is required
- Do not change needle prior to inoculation into culture bottles

- Inoculate Blood Culture Bottles with appropriate volume of blood as given on the bottle label.

a. Clean site with 70% alcohol to remove iodine
b. Allow to completely dry before injecting
c. Use a sterile needle an aseptic technique during ver: puncture
d. Inject blood immediately into bottle with same needle: do not touch needle

ii. Blood cultures are best taken by venipuncture, next best alternative is to take sample from the arterial line

iii. Skin organisms grown from a single bottle are usually considered a contaminant but shall be interpreted in the context of the patient

4. Investigation of pneumonia
   a. Community acquired pneumonia
      i. Usual organisms: Strept, pneumoniae, H.influenza
      ii. Less commonly
      a. Bacterial: legionella sp., Gram – negative bacilli, Staphaureus
      b. Viral: Influenza A, B, Parainfluenza, Adenovirus, RSV

c. Others Mycoplasma pneumoniae
Chlamydia psittaci (birds)
Coxiella burnetti (sheep or cattle)
Mycobacteria (TB and NTM or MOTT), Chlamydia Pneumoniae

iii. Investigation

a. Hematology – High (more than 15000/mm^3) or Low (less than 3000/mm^3) WCC - coagulopathy
b. Biochemistry – note Renal Function and LFTs
c. CxR
d. ABGs
e. Microbiology: *prior to antibiotic treatment where possible
1. Blood cultures x 2 sets

2. Endotrachel aspirate
i. KOH Mounts, CandS and urgent gram stain
ii. Legionella culture**
iii. Respiratory viral Ag**

NB: if not intubated, then collect a nasopharyngeal aspirate for respiratory viruses**

3. Serology – Legionella**, Mycoplasma, Respiratory viruses**
4. Urine – L pneumoniae – 1 Ag
5. Pleural fluid – KOH Mount, gram staining, AFB staining, Aerobic, Fungal and AFB Cands

b. Community acquired pneumonia – Immunosuppressed Host

i. Possible organisms

a. As above plus
1. Bacterial: Nocardia
2. Viral: CMV, HSV, Varicella Zoster
3. Fungal: Candida, Cryptococcus, Aspergillus
4. Protozoal: Pneumocystis carinii

ii. Investigations: *As per above, plus
a. Sputum or tracheal aspirate for
1. Fungal stain and culture
2. Silver methanamine stain/ DFAT for Pneumocystis. HIV patients only, all others require BAL

iii. Treatment prior to microbiological diagnosis: refer to antibiotic / antimicrobial guidelines

c. Nosocomial pneumonia in ICU
i. Principles
a. Accurate diagnosis and appropriate treatment are important but difficult

a. Incidence: 20% of all ICU patient 70% of patients with ARDS, major cause of death in patients with ARDS
b. Clinically indistinguishable from pulmonary fibrosis, alveolar hemorrhage, atelectasis and other causes of lung infiltrates
c. Clinical diagnosis, including use of tracheal aspirate, has poor sensitivity and specificity
d. Appropriate antibiotics do improve outcome, empiric broad – spectrum antibiotics are potentially harmful

ii. Consider nosocomial pneumonia when

a. New and persistent CxR changes
b. Tachycardia, tachypnoea
c. Fever or hypothermia – temperature more than 37.5 or less than 35.5 degrees C
a. Leucocytosis or leucopaenia – WCC: more than 11 or less than 4 x 109/1
b. Purulent sputum
c. Deterioration in lung function

iii. Confirmation of pulmonary infection:

a. Tracheal aspirate with **quantitative culture** is the most practical method of diagnosing nosocomial pneumonia. Quantitative culture
b. Sensitivity and specificity may be comparable to bronchoalveolar lavage and protected brush specimens in many situations
a. Preliminary results to direct therapy may be obtained on gram stain
b. Consideration shall be given to obtaining pulmonary samples by bronchoalveolar lavage (or open lung biopsy) for patients with

a. Persistent sings of pneumonia
b. Inadequate response to antibiotics
c. Inability to obtain adequate tracheal aspirates, or

d. To exclude non-infectious causes of respiratory failure (for example, interstitial fibrosis or alveolar haemorrhage)

c. Maximum value that can be elicited out of BAL for diagnosis of Nosocomial pneumonia can be only if the patient has been antibiotic free for more than 48 hours
Refer to BAL in procedure section
d. Septic screen including blood cultures shall be performed

5. Vascular catheter sepsis

a. Refer to the invasive procedure section
b. Suspect line sepsis when

i. Evidence of systemic infection

a. New, unexplained fever

b. Deterioration in organ function

c. Positive blood culture by venipuncture with likely organisms (Coagulase negative Staph, Candida)

ii. Evidence of local infection – inflammation or pus at the insertion site

iii. The following patients are more susceptible to line infections:

a. Prolonged vascular access (more than 7-10 days). The incidence of line infection increases exponentially after 4 days
b. Endovascular infection (SBE, prosthetic graft infection)
c. Cutaneous infection
d. Burns
e. Severe intra-abdominal infection (Pancreatitis) or deep seated infections (emphyaemia/abscess)

iv. In case of line sepsis with antibiotic impregnated lines (incidence of line sepsis is around 1%), the most likely organisms include Candida sp.

c. Protocol

i. Take blood cultures from two different peripheral venous sites
ii. Positive bottles shall be carefully labeled as to the site of sampling

a. Organisms commonly implicated in line sepsis are hose that normally colonize the skin: Staph.Sp., C. albicans, but in the ICU setting low pathogenicity potential gram negative organisms (Chryseobacterium, Burkholderia, Stenotrophomonas etc) and other gram-negative bacilli can also be involved

iii. On suspicion of line sepsis the line shall be removed

iv. The tip of the catheter shall be sent for culture. To avoid contamination of the catheter tip with skin organisms the skin shall be cleaned thoroughly with alcohol, allowing at least one-minute drying time, before removing the catheter

v. Catheter related bloodstream infection is defined as infection where the same organism is grown from the blood and from the catheter tip

d. Treatment

i. Removal of the infected line shall usually clear the bacteraemia and result in a fall in temperature and resolution of septic signs.

ii. Antibiotics are only indicated if signs of sepsis continue after the catheter has been removed, or the patient is a high risk patient (for example, prosthetic implants)

iii. If this occurs, more blood cultures shall be taken prior to starting antibiotics

iv. Refer to antibiotics guidelines

e. Further venous access

i. In ICU, as central access is usually required for inotropes or TPN, removal of a line usually involves reinsertion of another

ii. If possible wait 24 hours before reinsertion

iii. Whenever possible insert a new line at a different site

iv. Guide wire exchanges are not performed unless

a. Mechanical problems in a new catheter (leaks/kink and less than 4 days old)

b. Difficult or limited central access (for example, burns) Mechanical problems in a new catheter (leaks/kink and less than 4 days old)

c. Difficult or limited central access (for example, burns) Mechanical problems in a new catheter (leaks/kink and less than 4 days old)

d. Difficult or limited central access (for example, burns) Mechanical problems in a new catheter (leaks/kink and less than 4 days old)
e. Difficult or limited central access (for example, burns)

6. Bacterial Meningitis

a. Antibiotics are as per the requirement, which is case dependent. Refer to antimicrobial guidelines

7. Fungal infections

a. General principles

i. The incidence of systemic fungal infections in Intensive Care patients has increased in recent years
ii. This is due to increased numbers of immunosuppressed patients admitted to ICU, the use of broad-spectrum antibiotics and prolonged use of intravascular catheters
iii. Fungaemia is generally used as an indication to commence antifungal therapy. Whilst Candidaemia is associated with

significant morality, systemic infections can occur even when blood cultures are negative.s

b. Risk factors for Candidaemia and disseminated Candidiasis

i. Neutropaenia
ii. Long term CVC use
iii. Candida colonization
iv. Broad Spectrum antibiotics
v. Haemodialysis
vi. Immunosuppressants

c. Antifungal prophylaxis

i. Systemic prophylaxis is not recommended for general ICU patients
ii. Solid organ transplant patients do not require prophylaxis
iii. Fluconazole prophylaxis is effective and tolerated in bone marrow transplant and neutropaenic therapy

d. Indications for antifungal therapy

i. Candiduria in a high-risk defined patient (debilitating disease, uncontrolled diabetes, prolonged broad spectrum antimicrobial therapy with deteriorating clinical status
ii. Single positive blood culture in a high-risk patient
iii. Isolation of Candida from any sterile body site (except urine)
iv. Positive microscopy for yeast from a sterile specimen
v. Histological evidence of yeast or Mycelia forms in tissue from high-risk patients
vi. Fungal susceptibility testing for yeasts shall be asked for in all such cases as soon as the results of positive fungal growth are available

Specific antifungal therapy

8. Protocol for patients with narcotizing soft tissue infections

a. General principles

i. This is a generic group of patients with life threatening infections involving combinations of Mucocutaneous, fascial and myofascial planes
ii. These infections represent a medical emergency patient may present with severe septic shock and rapidly developing multiple organ failure

iii. In rapidly progressive infections, local signs of inflammation may underestimate the degree of underlying tissue necrosis
iv. These infections are usually due to one or more the following organisms

a. Anaerobes (Clostridium sp, Bacteroides)
b. Gram positives (Group A Streptococcus, Staphylococcus aureus)
c. Gram negative (Enteric organisms, Pseudomonas aeruginosa)

b. Management protocol

i. The hallmarks of management involve a detailed multidisciplinary approach coordinate by the duty Intensive Care consultant and involving the following
ii. Prompt and effective resuscitation, restoration of vital organ perfusion and control of metabolic emergencies
iii. Send appropriate specimens for Microbiology, before starting antimicrobials
iv. Hit and hit hard in critical situations
v. Prompt identification antibiotics / antimicrobials as required
vi. De escalate as soon as possible
vii. For usage of restricted antimicrobials, Sr.Consultant, Microbiology /

Infection Control Team shall be notified as soon as possible

1. Indications for hyperbaric oxygen on Microbiological grounds is – Progressive bacterial (Clostridial) gas gangrene

*Only where high index of suspicion or outbreak;

APPENDIX

Collection of Microbiology specimens

Urine culture

i. The Timing of Collection

Best samples are obtained before the administration of antimicrobial therapy. If already on antimicrobials, preferably at least 72 hrs, gap shall be there between the last dose administrated and the sample collected Day's first voided samples are preferred. If it is not possible, then the patient is asked to hold back voiding urine for as long as possible i.e., the total time between the last void and the sample has to be maximized. Intake of large volumes of fluids shall be avoided before the sample is obtained

ii. Methods of collection

i. MSU (Mid Stream Urine)
Adequate periurethral washing before samplying yields a fairly good sample
Washing with any good soap solution and water or even copious amounts of water along can be used
The hands of the collector shall also be washed. The patient is instructed to sit on the toilet seat

Female patients

- The female patient shall sit with her knees widely separated. The labia are separated, urethral meatus is cleaned atleast 4-5 times with recommended soda soaked gauze sponge or with plenty of water only is soap is unavailable
- A front to back stroke (downward) is a must, discarding the gauze after each stoke

- The urethral meatus is then rinsed with gauze sponges moistened with water in the same manner as with soap
- Keeping the labia separated, the patient shall void about 15-30 ml (initial void) into the toilet seat and without any stopping of the urinary stream, the specimen (about 15-20ml) is collected in a sterile container
- The rest is again voided into the toilet

Male patients

- In males, the foreskin or the prepuce has to be retracted in the uncircumcised patient
- The urethral meatus is adequately washed with soap and then water or with plenty of water only if soap is not available
- Urine sample is then taken as above

2. Sample from Catheter (Chronic Indwelling Urethral Catheter)

Sample form such catheters usually do not reflect pathogen

The catheter tubing shall be clamped away from the sampling area, which shall be as near to the urethral meatus as is possible (to collect freshly voided sample). The soft rubber connecter between catheter and collecting tubing can also be utilized for this purpose.

The site of sampling shall be prepared as for I/V line, with povidone-iodine and then 70% alcohol. The urine is collected by aspiration from the prepared site with sterile needle No. 26/28 and syringes, and then the samples is quickly transferred to a sterile container

Sample shall NEVER be collected from the collection bag or disconnecting catheter from drainage bag or from the urine pot

3. Heal Conduit / Urostomy Specimen

Strict aseptic precautions are required for collection of sample

The opening shall be cleansed and specimen collected through a double lumened catheter inserted beyond the fascial level

Alternatively a fresh urostomy bag is used to collect sample after cleansing the opening

4. Clean Initial Void Urine

For diagnosing urethritis, after proper cleansing, as described above for MSU sample, the early stream (5-10) ml and late stream samples from single voiding are taken into consideration for culture. Both the samples shall be sent as separate urine cultures indicating clearly the initial and the late stream sample on the request form/ requisition slip and label on the container

5. Segmented Urine Sample

For the diagnosis of prostates, sequential specimens are collected along with prostatic massage

The initial 5-10 ml of voided urine, then MSU sample, followed by prostatic massage and collection of expressed out prostatic secretion and finally post prostatic urine (2-3 ml of post massage voided urine) form the samples for culture. All the samples shall be sent as
separate culture indicating clearly the initial / MSU / prostatic massage / post prostatic massage urine on the accompanying request form/ requisition slip and label on the container

6. Suprapubic Aspiration

After appropriate and proper preparation of the part and with total aseptic technique, at least 1.0 ml of urine shall be aspirated for culture of aerobes and anaerobes. Larger volumes are required for Mycobateria and Fungi

The sample can be sent either in the appropriate BacTAlert and / or Anaerobic (Pediatric) bottle and quickly sent to Microbiology Laboratory in a sterile container especially if gram staining or other microscopy is required

Suprapubic aspiration is recommended in

a. Diagnosis of bacteriuria in infants and small children
b. For evaluating repeatedly doubtful counts of low count clean voided MSU
c. When anaerobic organisms are suspected to be the causative agents

Contraindicated in Coagulopathies

7. Sample of Mycobacteria

First voided whole early morning sample shall be collected in a sterile container obtain form the Department of Microbiology and submitted on three consecutive days with a separate request for each day. Both AFB staining as well as AFB culture shall be asked for

8. Sample for Anaerobic Bacterial Culture

Suprapubic aspiration is the recommended method

A prior intimation and interaction with the Microbiology laboratory is necessary for such culture

At least 1.0 ml of urine shall be aspirated, optimally 5.0 ml to 10.0 ml. The urine shall be sent immediately to the laboratory in the syringe in which it was aspirated. Any residual air shall be expelled out form the syringe and preferably a rubber stopper is to be applied. Alternatively the sample can be sent in an appropriate BacTAlert Culture Bottle

9. Sample in Pediatric and Neonatal Age Group

Following samples are usually provided

- Invasive procedure

1) Supra pubic aspirate (Recommended with strict aseptic precautions)
2) In and out catheterization

- Non-Invasive Methods
    1) Supra pubic tapping
    2) The area is cleaned as for MSU about an hour after feed
    3) Baby is tapped just above the pubis with two fingers
    4) 1tap / second is given for 1 minute (60 taps / min)

This is following by rest for 1 minute and the cycle repeated

MSU (if possible) as above

10. External Collection Devices

Often used in neonates and males with urinary incontinence

Due to the proximity to skin and urethra the urine collected may be unreliable for determining bladder bacteriuria.

Repeated growth of the same uropathologents may help in such cases if initially the bag has been aseptically applied

URINE SPECIMENS UNSUITABLE FOR MICROBIOLOGICAL PROCESSING

- Specimens obtained from urinals / bedpans
- Specimens mixed with stool
- Midstream Urine sample for Anaerobic Culture
- Urine collected from catheter bags directly
- Diluted specimens
- Specimens collected in leaking / broken containers
- Specimens contaminated by foreign materials like dyes, oily chemicals etc.,
- Specimens left at room temperature without any preservation for more than 2 hours.

- Frozen samples

Samples not be encouraged

Tips of Foley's catheters, until and unless justified

Blood culture

Blood culture bottles

Different Culture Bottles (BacTAlert) are available for different types of organisms i.e.,

For Aerobic	:	BACTEC PLUS+ Or	Aerobic / F
			BacT/ ALERT FN
For Fungal	:	Myco / F Lytic / Mycosis	
For AFB / Mycobacteria	:	Myco / F Lytic	

Collection procedure: follow as given above

Sputum

Well and deep coughed out sputum sample is required. The sample shall be coughed out directly into the sterile container and shall not be mixed with saliva

Postural drainage or help from physiotherapists or respiratory consultants may be required to stimulate coughed out sputum in cases where spontaneous output is lacking

Supervised (under a respiratory consultant) aerosol – induced sputum may also be given in such cases where it is not contraindicated

It shall be immediately transported to Microbiology Department

Urethral Swab

After taking proper universal precautions, a swab is inserted 2 cm inside the urethra and rotated gently before withdrawing back. Separate swabs are required for gram's staining and for culture for Gonococci. If other organisms are also being suspected it is advisable to send as many swabs. Special media (Stuart) is required for transportation of these swabs in case of delay. This is available from department of Microbiology on demand.

It shall be immediately transported to Microbiology Department

Elisa and Serological tests

About 5-10 ml of aseptically out blood collected in sterile red capped plain vacutainer tube or equivalent plain tube without any additives is required for ELISA tests.

Fasting samples are generally not required until and unless specified so.

Sample for the following tests are drawn by Microbiology Laboratory Technical Staff after receipt of requisition slip in the Microbiology Lab.

- Cryoglobulins
- Cold Agglutinins

All samples especially for Complement component shall be sent as quickly as they drawn out.

INSULIN INFUSION

1. Indications for use

1.1 The patient populations which may benefit from this protocol include, but are not limited to:

- Ischemic stroke
- ICH
- SAH
- Traumatic Brain injury
- Status Epilepticus
- Menningitis/Encephalitis
- Mg+
- GBS

1.2 Patients must have documented hyperglycemia as defined by:

1.2.1 2 or more glucose levels of greater than 150 mg/dl
1.2.2. Admission glucose > 250 mg/dl

1.3 Protocol will be implemented when an RD/Consultant order is written on the case file.

1.4 Exclutions

- Blood Glucose > 300 mg
- Diabetic ketoacidosis
- Hyperosmolar Non-ketotic Coma
- In patients in whom testing of glucose using a glucometer is in appropriate and do not have a central or an arterial line for the frequent monitoring of the blood sugars.

2. Responsibility

2.1 RD/Consultant

i. Assess patients need to be on the insulin infusion for hyperglycemia
ii. Enter the order on the case file.

iii. Asses if the insulin drip protocol is appropriate.
iv. Discontinue the insulin infusion as appropriate while transporting for small procedures, and 12 hours prior to the transfer to ward/room.

2.2 Nurse

2.2.1. Will monitor the blood sugars as per the protocol.
2.2.2. Titrate the infusion as per the protocol.
2.2.3. Documentation of blood sugars and insulin infusion rate.

3. Assessment

3.1 RD/Consultant

3.1.1. Assess the factors that may increase or decrease the insulin requirements
i. Assess the electrolytes requirements as per the protocol during the infusion
ii. Assess the signs and symptoms of hypoglycemia and to restore normoglycaemia.
iii. Assess the adequacy of the blood sugar control and to alert the ICU Coordinator when the blood sugar levels fall outside the range between 100 to 180 mg for two hours consecutively.

Nurse

3.2.1. Assess the glucose levels as per the protocol and decide the insulin requirement
3.2.2. Assess for the signs and symptoms of hypoglycemia and inform the ICU coordinator/The treating physician.
3.2.3. Assess the adequacy of the blood sugar control and to alert the resident Doctor when the blood sugar levels fall outside the range

between 100 to 180 mg for two hours consecutively.

3.2.4. Assess patients for factors that may influence the accuracy of the glucometer reading such as peripheral disease, anaemia, polycythemia, dehydration, shock, severe edema or hypotension.

3.2.5. If a patient has any of the above factors that would affect the accuracy of the glucometer reading, the sample must be drawn from the arterial or a central venous line and performed on a glucometer.

4. Interventions

4.1 50 units of Human Actrapid is diluted in 50 mls of normal saline loaded on a syringe pump set at the rate as per the protocol.

4.2 Dextrose normal saline is infused at a rate of 100ml/hr only with help of an infusion pump or dorsiflow.

4.3 Insulin is compatible with many other drugs and it does not need a dedicated line.

4.4 If the patient meet any of the following criteria, send blood specimen to the lab.

Proceed with adjustments to the insulin drip per protocol until confirmation from lab.

- Blood glucose >400 or <60 via bed side testing.
- Hematocrit is <20 or >55, send blood specimen to lab
- Patient meets any of the criteria listed in 3.2.4

4.5 Blood Glucose Monitoring

4.5.1 Check blood glucose hourly until stable (three consecutive values with in the target range)

4.5.2 Then check every q 2 hours

4.5.3 If any of the following occur, consider the temporary resumption of the hourly blood glucose until stable.

- Any change in the insulin infusion rate (blood glucose outside the target range)
- Significant change in the patient clinical condition.
- Initiation or cessation of the vasopressor or steroid therapy
- Initiation or cessation of renal replacement therapies.
- Initiation or cessation or rate change of nutritional supports, TPN, EN, PPN

5 Reportable conditions

5.1 Blood Glucose level <60
5.2 Blood Glucose level > 300
5.3 Signs and symptoms of Hypoglycaemia 5.4 Inadequate response to therapy. (Drip rate more than 25 units / hour)

6. Documentation

6.1.1. Blood glucose levels
6.1.2. Insulin drip rate
6.1.3. Implementation of the protocol

INSULIN DRIP

1. Target blood glucose level 80 to 160mg
2. Initial blood sugar scales to be followed as per the instructions given by the Endocrinologist / ICU Coordinator.

3. The blood sugars of the patient outside the target range for two consecutive hours to be informed to the endocrinologist / ICU coordinator

Table: INSULIN SCALES

Scale A		Scale B		Scale C	
	Units		Units		Units
< 100	STOP	< 80	STOP	< 80	STOP
101- 120	1	80- 100	0.5	80- 100	1
121- 140	2	101- 120	1	101- 120	2
141- 160	3	121- 140	2	121- 140	4
161 -180	4	141- 160	4	141- 160	6
181- 200	5	161 -180	5	161 -180	7
201- 220	6	181- 200	6	181- 200	8
221- 240	7	201- 220	7	201- 220	9
241- 260	8	221- 240	8	221- 240	10
261- 280	9	241- 260	9	241- 260	11
281- 300	10	261- 280	10	261- 280	12
301- 320	11	281- 300	11	281- 300	13
321- 340	12	301- 320	12	301- 320	14
341- 360	13	321- 340	13	321- 340	15
361- 380	14	341- 360	14	341- 360	16
>380	16	361- 380	15	361- 380	19
		>380	16	>380	20

10. Neurology Protocol

ICU MANAGEMENT OF STROKE

INITIAL MANAGEMENT OF STROKE

- AIRWAY
- BREATHING
- CIRCULATION

POSSIBLE COMPLICATIONS

- Increased ICP
    - Cerebral Edema
    - Hydrocephalus
- Seizures
- Hemodynamic Instability
- Arrhythmias
- Deep Vein Thrombosis (DVT)
- Pulmonary Embolus (PE)
- Hyperglycemia
- Infection
- Electrolyte Abnormalities
- Pain

- Confusion/Delirium

INCREASED INTRACRANIAL PRESSURE

- In ICU q 1 hr x 8, then q 2 hr
- Exam includes:
  - GCS, Cranial Nerves, Motor
- Post rtPA:
  - In ICU
  - Every 15 minutes during infusion
  - Every 30 minutes x 6 hours
  - Every hour x 24 hours from treatment

Cerebral Edema

- Seen with large multilobar infarcts
- Risk within first 24 hours with cerebellar infarct
- Peaks at 3-5 days after infarct
- Major contributor of mortality after intracerebral hemorrhage (ICH)

Management Objectives Of raised ICP

- Decrease ICP
- Maintain adequate cerebral perfusion pressure
- Prevent herniation

Goals

- ICP < 20 mmHg
- CPP > 70 mmHg

MANNITOL

Mannitol 0.25-0.5 mg/kg IV over 20 mins

- Can be given q 4-6 hours
- Max daily dose 2g/kg
- Not longer than 5 days
- Goal serum osmolality < 310
- CAUTION: After prolonged administration (continuous infusion) may exacerbate cerebral edema and raise ICP.

HYPERVENTILATION

- Reducing CO2 b 5-10 mmHg decreases ICP by 25-35%
- Co2 to be reduced to 30 to 35 mmHg and never less than 35 mmHg
- Short term, temporizing measures since ongoing vasoconstriction exacerbates ischemia

Hydrocephalus

Due to,
- IVH
- Compression

Placement of extraventricular drainage device

Surgical Intervention

- Recommended for decompression of large CEREBELLAR infarctions causing brain stem compression and hydrocephalus.
- Large hemispheric infarct: surgical decompression and evacuation can be life-saving measure, but survivors have severe residual neurological impairments.

HYPERTENSION : ISCHEMIC STORKE

- Permissive HTN
  > Not eligible for tPA
    - § SBP ≤ 220mmHg/ or DBP≤120mmHg non-tPA
  > Eligible for tPA
    - § Pre Rx: SBP < 185 or DBP < 110
    - § Post Rx: SBP < 180 or DBP <1

HYPERTENSION: HEMORRHAGIC STROKE

- § PMHx HTN = MAP < 130 (unless post-op, then MAP < 110)
- § SBP < 180/ DBP < 110
- § CPP > 70

TREATMENT

- Volume first: isotonic, colloid
- Vasopressors
  - Phenylephrine
  - Norepinephrine
  - Dopamine

SEIZURES

- **HEMORRHAGIC STROKE**
  - o Prophylactic administration of antiepileptic drugs (AEDs) may be considered for up to 1 month and then discontinued if no seizure
  - o Evidence supporting this is lacking

- **ISCHEMIC STROKE**
  - o Low incidence
  - o Most likely to occur n 1st 24 hours
  - o No data to support PROPHYLACTIC administration AEDs
  - o Therefore, treat only if have a seizure

GLYCEMIC CONTROL

- Euglycemia is the goal
    - 80-110 mg/dl

- Strict sliding scale insulin
    - Low dose
    - High dose

- Appropriate diet
- Insulin drip*

TEMPERATURE MANAGEMENT

Normothermia Protocol

- Keep temp < 37.5 rectally
- Acitiominophen
- Cooling blanket
- Surveillance Cultures q day/q o day
- Hypothermia: not enough data to recommend use for stroke

DEEP VEIN THROMBOSIS

- Anti-embolism stockings
- Sequential compression devices
- Early mobilization
- Subcutaneous administration of heparin if no contraindication.

Adult Intracerebral Hemorrhage

1. Guidelines for Emergency Management of Intracerebral Hemorrhage

Identification of patients with suspected intracranial hemorrhage requires urgent brain imaging. Unenhanced CT is the study of choice given its availability, ease of use and sensitivity to subarachnoid hemorrhage, but MR imaging may contribute to the evaluation and management of suspected brain hemorrhage. Intracranial hemorrhage includes epidural (EDH), subdural (SDH), subarachnoid (SAH), intraventricular (IVH), hemorrhagic transformation of ischemic stroke (HT), venous hemorrhage from cortical vein or sinus thrombosis (CVST), and intracerebral (ICH). For patients with ICH, the following underlying conditions must always be considered: coagulopathy, trauma, vascular lesions, venous thrombosis, aneurismal rupture and hemorrhagic mass lesions such as tumors.

2. The Following Guidelines Apply to Intracerebral Hemorrhage (ICH)

These guidelines should be used only as medical and educational reference tools. They are not intended to be used as a diagnostic decision-making system and must not be used to replace or overrule a physician's judgement or diagnosis. Application of this information in a particular situation remains the professional responsibility of the practitioner.

The following steps should be considered in parallel rather than in sequence, especially in the stabilization of vital functions and correction of coagulopathy.

1) Assess vital functions. Determine if intubation is required for patient safety during imaging evaluation. If so, consider use of an ultra-short acting neuromuscular blockade or sedative-hypnotics agent to allow for rapid return of motor control and assessment of neurologic deficits. Establish if co-morbid acute myocardial injury is a risk in patients with severely elevated BP.

2) STAT PT/INR, PTT, CBC with platelets, D-dimer, fibrinogen, electrolytes, BUN/Cr, glucose, liver function tests, type and screen to blood bank.

3) Alert neurosurgery. Cerebellar ICH is a neurosurgical emergency. Hematoma evacuation can be considered for patients with lobar ICH who demonstrate progressive clinical deterioration. Patients may also be candidates for intracranial pressure monitoring or emergency external ventricular drain placement.

4) Consider the Different diagnosis:

- Spontaneous ICH (SICH): Unless there is a contraindication to contrast use, such as renal failure or contrast allergy, vascular imaging (typically CT angiography) is usually indicated to assess for underlying aneurysm or vascular malformation. Page the acute stroke team for evaluation for clinical trial eligibility (Beeper 34CVA).
- If an aneurysm is confirmed, contact neurosurgery immediately and see the subarachnoid hemorrhage protocol .
- ICH secondary to underlying tumor, AVM cavernous malformation, and venous sinus thrombosis: Additional brain imaging is necessary to exclude an underlying lesion. Both contrast CT and contrast MRI may be useful initially and several weeks later once hemorrhage products have begun to be reabsorbed. Digital subtraction angiography can also be useful in certain settings.
- Hemorrhagic conversion of ischemic infarction (HT): Additional brain imaging with MRI (and DWI if available) may be needed to confirm the underlying ischemic etiology in regions that were not subject to hemorrhagic transformation. Regions of subtle petechial hemorrhage that are not visible on unenhanced CT but easily seen on MR gradient echo susceptibility sequences have an unclear significance with respect to initiation of antithrombotic therapy. If acute ischemic stroke is suspected (onset less than 12 hours), contact acute stroke team immediately (Beeper 34CVA).

5. Measure ICH volume, GCS, NIHSS. Measure volume using ABC/2 method, where A is the greatest hemorrhage diameter by CT, B is the diameter 90

degrees to A to C is the approximate number of CT slices with hemorrhage multiplied by slice thickness in cm.

6. Correct coagulopathy
   - Warfarin

     - Immediate therapy

1. If patient is on warfarin and INR is elevated or if PT is elevated in the absence of warfarin therapy: administer Vitamin K 10 mg IV over 10 minutes followed by Fresh Frozen Plasma 10-20mL/kg (Each unit of FFP contains roughly 200 ml).

2. Vitamin K and FFP must be dosed STAT and the team must designate a single physician to take personal responsibility for ensuring that these therapies are administered as fast as possible.

§ Vitamin K should be administered within 5 minutes of the order.

§ As soon as FFP is ordered, a "runner" should be dispatched to the blood bank to collect FFP, which should be administered as soon as possible.

3. Notes on Warfarin reversal:

a. Intravenous vitamin K is associated with a small risk of severe allergic reaction. When administered intravenously, the rate should not exceed 1mg/minute. Reversal of anticoagulation by any means (vitamins K or FFP) is associated with a risk of thrombosis depending upon the patient's underlying indication for anticoagulation.

b. FFP should be dosed based on patient weight 10-20mL/kg (usual adult dose is 4-5 units). Dosing at the higher end is often necessary depending upon the initial INR. If FFP is administered without concomitant vitamin K, the effect of FFP will dissipate in 6-8 hours and so FFP should never be used without concomitant vitamin K for ICH. Depending on the clinical condition of the patient judicious administration of diuretics may be indicated.

Check for OTHER causes of coagulopathy:

- § Patients taking aspirin in addition to warfarin can be considered for platelet transfusion

- § For patients taking other antithrombotic agents, in addition to warfarin (ticlopidine, low-molecular weight heparins, etc), phone consult with the blood bank fellow on call, the acute stroke attending, or hematology.

Patients with anticoagulant-related ICH are at high risk for prolonged bleeding and hematoma expansion. Non-contrast cranial CT scan should therefore be repeated every 12 plus or minus 2 hours from time of initial CT scan until ICH volume is stable on greater than or equal to 2

Consecutive CT scans. In addition, CT scanning should be repeated when neurologic deterioration occurs.

Follow-up therapy

STAT PT/INR q 4 hrs x 24; then q 6 hrs x 36; then as needed.

If the INR is greater than 1.3 to 4 hours, administer second dose of Vitamin K 10 mg IV and infuse a second dose of FFP (10-20 ml/kg). Evaluate the patient for disseminated intravascular coagulation and phone consult a staff member of neurology, transfusion medicine or hematology. The Blood Bank attending or fellow on call should be paged to discuss whether the use of

alternative agents such as prothrombin complex concentrate should be considered.

- Standard (Unfractionated) Heparin

Immediate therapy: Discontinue the heparin infusion and order Protamine sulfate. Calculate total amount of heparin received over the preceding 3 hours.

If initiated with 30 minutes of last heparin dose: Give 1 mg protamine per 100U heparin

If initiated with 30-60 minutes: Give 0.5-0.75 mg protamine per 100U heparin

If initiated with 60-120 minutes: Give 0.375-0.5 mg protamine per 100U heparin

If heparin stopped greater than 120 minutes ago: Give 0.25-0.375 mg protamine per 100U heparin

Give by slow IV injection, not to exceed 5mg/min, with total dose not to exceed 50mg

Monitor for signs of anaphylaxis; the risk is higher in diabetics who have received insulin.

Follow-up therapy

STAT PTT q1 hour for the next 4 hours, then q4 hours through 12 hours of hospitalization.

- Low Molecular Weight Heparin—Protamine sulfate reverses only about 60% of the anti-factor Xa activity of low-molecular-weight heparin, has negligible effects on danaparoid (a mixture of anticoagulant glycosaminoglycans used to treat heparin-induced thrombocytopenia) and fondaparinux (a synthetic antithrombin-binding pentasaccharide with exclusive anti-factor Xa activity)

Enoxaparin: 1mg protamine for each mg of enoxaparin; if PTT prolonged 2-4 hours after first dose, consider additional dose of 0.5 mg for each mg of enoxaparin.

Dalteparin or tinzaparin: 1 mg protamine for each 100 anti-Xa IU of dalteparin or tinzaparin; if PTT prolonged 2-4 hours after first dose, consider additional dose of 0.5 mg for each 100 anti-Xa IU of dalteparin or tinzaparin.

- Direct Thrombin Inhibitors (Argatroban, Lepirudin, Bivalirudin, Ximelagatran): There is no specific antidote for these drugs at this time. Consider antifibrionolytic agents such as Amicar (EACA). Phone consult with the blood bank fellow on call, the acute stroke attending, or hematology.

o Thrombolytic Agents

Consult Neurosurgery for possible intervention.

Check STAT labs: CBC, PT, PTT, platelets, fibrinogen and D-dimer

If fibrionogen, less than 100 mg/dL, then given Cryoprecipitate 0.15 units/kg rounded to the nearest integer. If still bleeding at 1 hr and fibrinogen level still less than 100 mg/dL, repeat cryoprecipitate dose.
Institute frequent neurochecks and therapy of acutely elevated ICP, as needed.

Additional Options or considerations

If platelet dysfunction suspected, give platelets 4 units
If heparin has been administrated in the past 3 hours
Discontinue the heparin infusion and order Protamine sulfate. Calculate total amount of heparin received over the preceding 3 hours.
If initiated within 30 minutes of last heparin dose: Give 1mg protamine per 100U heparin.
If initiated within 30-60 minutes: Give 0.5-0.75 mg protamine per 100 U heparin.
If initiated within 60-120 minutes ago: Give 0.25 to 0.375 mg protamine per 100U heparin.

Give by slow IV injection, not to exceed 5mg/min, with total dose not to exceed 50mg.
Monitor for signs of anaphylaxis; the risk is higher in diabetics who have received insulin.

Follow-up with STAT PTT q1 hour for the next 4 hours, then q4 hours through 12 hours of hospitalization.

For uncontrolled, life-threatening bleeding, consider aminocaproic acid (Amicar) 10g IV in 250 cc NS IV over 1 hr as a last resort. Note there is a significant risk of pathologic thrombosis with Amicar.

Serious systemic hemorrhage should be treated in a similar manner. Manually compress and compressible sites of bleeding, and consult appropriate additional services to consider mechanically occluding arterial or venous sources of medically uncontrollable bleeding.

- Platelet disorder

Antiplatelet agents, such as aspirin: Platelet transfusions are of uncertain benefit but can be considered.

Thrombocytopenia (platelet count less than 100,000/uL)—Transfuse with platelets until platelet count exceeds 100,000/uL

Von Willebrand syndromes: Treat with 0.3 mcg/kg DDAVP given IV over 30 minutes. Phone consult with a staff member of hematology or transfusion medicine for dosing of VWF factor concentrate.

DDAVP may also benefit patients with:

1. Uremic platelet dysfunction
2. Congenital platelet function disorders

3. Recent ingestion of combinations of antiplatelet agents such (e.g., ASA and clopidogrel)

7) Blood pressure management

a. Blood pressure should be managed according to American Heart Association 2007 Guidelines for the Management of Intracerebral Hemorrhage. All patients who require treatment with continuous intravenous antihypertensive therapy should undergo urgent placement of an intra-arterial catheter for blood pressure monitoring and central venous catheter for central venous pressure monitoring as well as administration of IV antihypertensive medications. Once a physician determines that a patient requires treatment with IV antihypertensive therapy, he/she must designate an individual who will remain at the bedside and monitor effectiveness of therapy until blood pressure is controlled.

b. Elevated blood pressure (suggested medications in approximate order of preference)

i. Labetalol: 5-100mg/hr by intermittent bolus doses of 10-40 mg or continuous drip (2-8 mg/min)
ii. Nicardipine: 5 mg/hr increased by 2.5 mg/hr q15 minutes to max 15 mg/hr
iii. Esmolol: 250 mcg/kg as a load; maintenance use, 25-300 mcg/kg/min
iv. Enalapril: 0.625-5mg IV Q6h
v. Hydralazine: 5-20 mg IV Q30min
vi. Nitroprusside: 0.1-10 mcg/kg/min

c. The following suggested algorithm is adapted from the AHA 2007 Guidelines for ICH:

i. If SBP is greater than 200 mmHg or MAP is greater than 150 mmHg, then consider aggressive reduction of blood pressure with continuous intravenous infusion, with frequent blood pressure monitoring every

5 minutes.

ii. If SBP is greater than 180 mmHg or MAP is greater than 130 mmHg and there is evidence of or suspicion of elevated ICP, then consider monitoring ICP and reducing blood pressure using intermittent or continuous intravenous medications to keep cerebral perfusion pressure greater than 60 to 80 mmHg.

iii. If SBP is greater than 180 mmHg or MAP is greater than 130 mmHg and there is not evidence of or suspicion of elevated ICP, then consider a modest reduction of blood pressure (eg. MAP of 100 mmHg or target blood pressure of 160/90mmHg) using intermittent or continuous intravenous medications to control blood pressure.

iv. Any clinical deterioration in association with reduction of BP should prompt reconsideration of ongoing BP management strategy.

d. Hypotension

i. The etiology of hypotension must be established. Volume replenishment is the first approach. Isotonic saline or colloids can be used and monitored with central venous pressure. If CVP is normal or elevated in the setting of hypotension, then a pulmonary artery catheter should be placed to monitor pulmonary artery pressures. If hypotension persists after correction of volume deficit, continuous infusions of vasopressors should be considered, particularly for low systolic blood pressure such as less than 90 mmHg.

- Phenylephrine: 2-10 mcg/kg/min
- Dopamine: 2-20 mcg/kg/min
- Norepinephrine: 0.05-0.2 mcg/kg/min

7) Glycemic control. For glucose greater than 140 mg/dl institute insulin therapy either in the form of sliding scale dose regimen or continuous IV drips.

8) Seizures. Anti-epileptic therapy should always be used for treatment of known seizures. Brief periods of prophylactic antiepileptic therapy are of uncertain benefit in patients with either lobar or deep hemispheric hemorrhage and can be considered.

9) Temperature—Maintain temperature less than or equal to 38 degrees using PO/PR acetaminophen 650 mg q6h. In the setting of poor airway control or if temperature remains elevated despite acetaminophen, consider external cooling.

10) Repeat neuro-imaging: Non-contrast cranial CT scan whenever concern for ongoing hemorrhage or hematoma expansion is raised or in the setting of clinical deterioration.

MANAGEMENT OF NEUROTRAUMA

INTRODUCTION

- Over the past 30 years, remarkable progress has been made in improving outcome from traumatic brain injury (TBI).

- Overall mortalities from severe TBI have dropped from in the range of 36% in the late 1980s into the low-to-middle teens at specialized TBI centers.
- Combination of many improvements in brain injury care, trauma systems, and critical care that has produced the decrease in mortality.

Primary and secondary insult's

- PRIMARY INSULTANT is the physical damage that occurs during the traumatic event. It results from shearing or direct damage to the parenchyma or as injury to tissue or vessels that result in hemorrhage and compression of the surrounding brain.
- Secondary insults are those processes occurring following the injury. They may be induced directly by the traumatic event or result from processes (sometimes iatrogenic) that follow later, or they may be caused by associated, extracerebral events

Secondary insults

- Cerebral edema
- Metabolic derangements
- Calcium toxicity
- Excitotoxic injury
- Apoptosis
- Hypotension
- Hypoxemia

What difference can we make?

- We can modify the secondary insults.
- We can not undo the primary injury effects.

Airway management (A & B)

- Airway control and assisted ventilation should be accomplished in all patients with a Glasgow Coma Scale (GCS) of at least 8
- TBI patient where facial or other injuries, aspiration, agitation, or other factors conspire against adequate ventilation, oxygenation, or airway control.
- In the hospital, Endotracheal intubation should be accomplished using a rapid sequence intubation (RSI) protocol
- Even though these patients may be comatose, they still will elevate their ICP to the stimulus of Endotracheal stimulation, sometimes to the point of herniation.
- Adequate analgesia and sedation are required during intubation
- In all cases, the Endotracheal position of the airway should be confirmed by return of carbon dioxide
- Ventilation should be targeted at maintaining a PaCO2 of approximately 35 mmHg
- Capnography be used whenever possible to prevent inadvertent hypo – or hyperventilation
- Hyperventilation should be delivered to patients only where intracranial hypertension is strongly suspected because of the presence of abnormal papillary size or reactivity (eg, anisocoria or bilaterally dilating or blown pupils), motor posturing, or progressive neurologic deterioration
- Post trauma it is recongnized that the cerebral blood flow decreases significantly
- Hyperventilation leads to extreme ischemia and hence is strongly discouraged except for cont. There is a strong evidence of support; even a single episode of hypotension or hypoxia especially during intubation leads to a poor neurological outcomes
- Every effort must be done to ensure a smooth intubation

HYDRATION

- For decades, it was a widely accepted tenet of TBI management to keep the patient dry by restricting fluids
- Now it is accepted that fluid restriction in TBI is extremely hazardous, and this practice has been condemned

What should be the optimal blood pressure?

CPP = MAP – ICP)

Overall, assuming a minimal acceptable CPP value of 50 mmHg and using an ICP of 20 mmHg in the equation, a MAP of 70 mmHg is suggested at the lower treatment threshold during resuscitation

MAP threshold of 90 mmHg has been suggested as a treatment option

Which fluid for resuscitation?

Isotonic fluids are the choice of fluids, Hypotonic fluids like ringers or dextrose fluid are discouraged

Hyper tonic solutions are findings a place in the control of ICH

Are inotropic agents required?

They may be needed temporarily, when fluid resuscitation is being accomplished, but the MAP remains unacceptably low

Alternatively, pressors may be needed supplmentally, when full volume resuscitation does not establish an adequate MAP

In general, alpha agonists are preferred (such as phenylephrine), although, in young patients, some selective beta activity also can be useful (i.e., with noradrenaline)

Principles of management

- The meticulous attention devoted to oxygenation, ventilation, and perfusion during resuscitation must continue in the intensive care unit.
- Ventilatory management decisions must take into account that high intrathoracic pressure may impede cerebral venous return and thereby raise ICP. Therefore, the lowest inspiratory and end expiratory pressures necessary to achieve adequate oxygenation and ventilation should be used.
- Maintain adequate intravascular volume and perfusion, avoid hyponatremia and hypoosmolarity, and provide nutrition.
- Hyperthermia should be avoided and treated aggressively.
- Seizures must also be treated aggressively, because they raise CBF and therefore ICP.
- The patient's upper body can be raised 15 to 30 degrees (without flexing the neck) to facilitate cerebral venous drainage

The neck should be maintained in the midline to prevent kinking of the jugular veins.

What is optimal CPP?

- The condition since qua non of ICP management is maintaining an adequate CPP.
- Although some authors have argued that it is necessary to maintain the CPP at a markedly supraphysiologic level (i.e, 70 to 90 mmHg)
- The general consensus is trending more toward levels of 50-60 mmHg
- Indeed, there is a growing argument that the elevation of CPP serves mainly as an extra wide margin of safety against hypotensive episodes

Barbiturate coma

- Pentobarbital sodium is loaded at 10mg/kg intravenously over 30 minutes, followed by an infusion of 5 mg/kg per hour for three hours then maintained at 1-3 mg/kg per hour, titrated to burst suppression on continuous bedside electroencephalogram (EEG).

Serum levels are followed, but clinical and electrophysiological endpoints direct dosage

The infusion is maintained until ICP control is satisfactory for 24 hours, then backed off by approximately 50% per day

High-dose pentobarbital has a strong tendency to produce hypontension, the ischemic consequences of which obviate the benefits of its ICP control (43-45). Very careful attention to volume status, frequently supplemented by a need for invasive monitoring and pressor use

STATUS EPILEPTICUS PROTOCOL

Status Epilepticus is a medical emergency. Outcome correlates with rapidity of treatment and response, and with underlying etiology. Protocols provide a useful outline and checklist for treating physicians, and outcomes have been shown to be better is centers that utilize written protocols for evaluation and treatment.

TABLE : STAUS EPELICTCUS PROTOCOL

INTERVENTIONS
Initial Rapid Assessment : Airway, Breathing , Circulation
Record Vital Signs
Monitor :Oxygen Saturation, EKG
Establish IV access and have bloods sent:

CBC, Lytes, BUN/Cr, Glucose, Ca,Mg,Po4, LFTS, AED levels,Toxicology Screen, ABG
THIAMINE, 100 MG IV(For Chronic ethonolic patients)
25% DEXTROSE, 50 CC IV (incase of hypoglycemia)
LORAZEPAM, 0.1 MG/KG IV (<2 MG/MIN)
Fosphenytoin, 20MG/KG IV (150 MG/MIN) or Phenytoin (20 MG/KG @ 50 MG/MIN)
Begin concurrently with benzodiazepine {above}
Monitor EKG, Check BP Q 120 sec
Fosphenytoin,Additional 10MG/KG IV (150 MG/MIN) or Phenytoin (10 MG/KG @ 50 MG/MIN)
Monitor EKG, Check BP Q 60 sec
Send Repeat Phenytoin level - 20 min after load
INTUBATE (If not done previously)
INITIATE EEG MONITORING

Options for next step include
MIDAZOLAM 0.2 MG/KG IV (loading dose)(Preferred if BP is unstable)
Titrate dose (0.1-0.4 mg/kg/hr) to stop electrographic and clinical seizures
Use fluid or pressor to support BP if needed
OR
PENTOBARBITAL, 5 MG/KG IV (loading dose) to obtain burst suppression on EEG
Use fluid to support BP if needed, Add Pressor only if fluids fails or not clinically advisable
OR
PROPOFOL 1-2 mg / kg load,210 mg/kg/hr maintenance drip to stop clinical and EEG seizures or maintain burst supression on EEG
Obtain CT SCAN (if clinically indicated)
Correct underlying cause of Status Epilepticus
Adjust the principal anticonvulsants to therapeutic effect
Taper Midazolam,Pentobarbital or Propofol after above complete

CONCURRENT EVALUATION:
History, Exam, Check Labs, STAT Head CT (MGH : 6-6760 BWH :7213)
Cool patient if febrile

Consider antibiotics & LP, especially if febrile or not known epileptic
Notes:

- Potential indications for urgent EEG or continuous EEG monitoring
- Question of non-epileptic status (pseudo status)
- Question of absence vs.complex partial status epilepticus
- Failure to regain normal consciouness within a reasonable time after apparent cessation of seizures activity
- Monitoring of anesthetic suppression of epileptic activity (burst-suppression interval)
- All requests for urgent EEG and monitoring should be discussed with Epilepsy Fellow on call (Page through Partners or 726-3311)

BRAIN DEATH DETERMINATION / APNEA TESTING

SUMMARY

Lack of cerebral blood flow secondary to traumatic injury or critical illness may be encountered in the intensive care unit (ICU). Brain death determination is most commonly confirmed by clinical neurologic examination in conjunction with a positive apnea test (lack of spontaneous respiratory efforts in the presence of an elevated $PaCO_2$) and requires independent brain death determinations by two licensed physicians. In specific clinical situations, confirmatory tests may be indicated

INTRODUCTION

By the Uniform Determination of Death Act, "death" is defined as either "(1) irreversible cessation of circulation and respiratory functions, or (2) irreversible cessation of all functions of the entire brain, including the brainstem…"[1] Brain death, defined as the absence of clinical brain function when the proximate cause is known and demonstrably irreversible, is commonly encountered in the ICU setting following severe traumatic brain

injury, aneurismal subarachnoid hemorrhage, blunt carotid injury, hypoxic-ischemic brain insults, fulminant hepatic failure, or severe hypperfusion.[2,3] Brain death occurs when intracranial pressure (ICP) exceeds cerebral perfusion pressure (CPP), resulting in cessation of cerebral blood flow and oxygen delivery. The determination of brain death has significant legal and

RECOMMENATIONS

- **Level 1**
- None

- **Level 2**

- Brain death will be confirmed by two physicians licensed by medical council of India
- The determination of brain death should be made by a combination of clinical neurologic examination and apnea. Confirmatory test may be performed at the discretion of the physicians involved
- Documentation of brain death should include the following information:

1. Etiology and irreversibility of the patient's coma and overall clinical condition
2. Absence of brainstem reflexes
3. Absence of motor response to pain
4. Absence of spontaneous respiration despite a **$PaCO_2 \geq$** 60 mmHg
5. Justification for and result of additional confirmatory test(s)
6. Findings of repeat neurologic examination

- Pre-xoygenation as well as correction of hypotension and metabolic acidosis should be performed to avoid complications during apnea testing

- **Level 3**

- None

Recognition of brain death under certain circumstances

(1) For legal and medical purposes, where respiratory and circulatory functions are maintained by artificial means of support so as to preclude a determination that these functions have ceased, the occurrence of death may be determined where there is the irreversible cessation of the functioning of the entire brain, including the brain stem, determined in accordance with this section.

(2) Determination of death pursuant to this section shall be made in accordance with currently accepted reasonable medical standards by two physicians licensed. One physician shall be the treating physician, and the other physician shall be a board-eligible or board-certified neurologist, neurosurgeon, internist, pediatrician, surgeon, or anesthesiologist.

CLINICAL NEUROLOGIC EXAMINATION

The clinical neurologic examination supplemented in appropriate clinical situations by performance of one or more confirmatory tests, remains the standard for the determination of brain death.[3,6,7] Declaration of brain death requires not only a careful clinical examination, but also:

- Establishment of the cause of coma
- Ascertainment of irreversibility
- Resolution of any misleading clinical neurologic signs
- Recognition of neuroimaging studies
- Performance of any confirmatory laboratory tests deemed necessary

A clinical neurologic examination to determine the presence of brain death can only proceed if the following four prerequisites have been met:

1. Clinical or neuroimaging evidence of an acute central nervous system (CNS) catastrophic that is compatible with the diagnosis of brain death.

- Typically, computed tomography (CT) of the brain demonstrates a catastrophic brain injury

- A normal CT scan should raise doubt as to the diagnosis of brain death and lead to further imaging studies.

2. Exclusion of complicating medical conditions that may confound clinical assessment such as:

- Severe electroyte, acid-base or endocrine disorders
- Refractory shock (systolic blood pressure < 90mmHg)
- Guillain-Barre syndrome
- "Locked-in" syndrome

- A consequence of destruction of the pons, typically due to basilar artery thrombosis, in which the patient cannot move the limbs, grimace, or swallow, but retains consciousness, voluntary blinking and vertical eye movements.

3. Absence of drug intoxication, poisoning, or neuromuscular blocking agents.

4. Absence of severe hypothermia, defined as a core temperature < 32° C (90° F)

- Pupillary response to light is lost at core temperature of 28° to 32° C
- Brainstem reflexes disappear when core temperature drops below 28° C

A comprehensive clinical neurologic examination includes documentation of the presence of coma, the absence of brainstem reflexes, and apnea. Each of these three components is described in further detail below

1. Coma or unresponsiveness

a. No cerebral motor response to pain in all extremities (nailbed pressure and supraorbital pressure)

2. Absence of brainstem reflexes

The examination of brainstem reflexes requires the assessment of reflex pathways in the mesencephalon, pons, and medulla oblongata. As brain death occurs, patients lose their brainstem reflexes in a rostral-to-caudal direction with the medullar oblongata reflexes may require several hours to develop

Pupils (CN II & III)

Round or oval pupils measuring 4 to 9 mm with no response to bright light

Ocular movement (CN III, VI & VIII)

No oculocephalic movements should be elicited by rapid turning of the head (performed only when no fracture or instability of the cervical spine is present)

No deviation of the eyes to cold caloric stimulation

i. Each tympanum should be irrigated with ice water after the head has been tilted 30 degrees
ii. Allow 1 minute after injection and at least 5 minutes between testing on each side
iii. The presence of clotted blood or cerumen within the external auditory canal may diminish the stimulatory response.
iv. There should be no tonic deviation toward the cold stimulus

- **Facial sensation and facial motor response** (CN V & VII)

- No corneal reflex to touch of the corneal edge by a swab
- No jaw reflex
- No grimacing to deep pressure on nail bed, supraorbital ridge, or temporomandibular joint

- **Pharyngeal and tracheal reflexes** (CN IX & X)

- No response to stimulation of the posterior pharynx with a tongue blade
- No cough response to bronchial suctioning (moving the Endotracheal tube back and forth may not be an adequate stimulus)

3. Apnea (see below)

APNEA TEST

Apnea must be demonstrated as part of any brain death declaration. Apneic diffusion oxygenation is the procedure most commonly utilized to maintain oxygenation during apnea testing. Preoxygenation eliminates the respiratory nitrogen stores, accelerates the transport of oxygen, and significantly decreases the risk of hypoxic complications during the trial.[7] The threshold of maximal stimulation of the respiratory centers in the medulla oblongata has been arbitrarily at partial pressure of arterial carbon dioxide ($PaCO_2$) of 60 mmHg. In patients with basline hypercarbia (such as in chronic obstructive pulmonary disease), the criteria are

modified to assume maximal stimulation at a $PaCO_2$ 20 mmHg above basline.[6-8] At these levels of hypercarbia, patients with an intact brainstem can be expected to demonstrate spontaneous respirations.

Once the patient has been found to have a clinical neurologic examination consistent with brain death, and assuming that there are no contraindications, an apnea test is performed. This test, by definition, is performed solely in patients who are critically ill with varying degrees of organ dysfunction. As a result, apnea testing is associated with a significant risk of complications including acidosis (63%), hypotension (24%), hypoxemia (12%), and cardiac arrhythmia (3%) most commonly due to inadequate preoxygenation and acidosis. Thus, contraindications to apnea testing include hemodynamic instability SBP < 90 mmHg), acute respiratory failure requiring high-level ventilatory support, and metabolic acidosis. If present, these abnormalities should be corrected before attempting an apnea test. By so doing, the risk of complications is significantly reduced. If an apnea test cannot be safely performed, one of the other confirmatory tests, most commonly technetium-99m cerebral blood flow imaging, should be utilized.

The apnea test is classically described as being performed after disconnecting the patient from the ventilator with 6 L/minute of oxygen being provided via the Endotracheal tube through a catheter placed at the carnia. With close observation of the patient's end-tidal carbon-dioxide waveform, however, apnea tests are more easily and safely performed with the patient on mechanical ventilation using a continuous positive airway pressure (CPAP) mode. In a patient with an intact brainstem, spontaneous respiratory efforts are most likely to develop early in the test as the patient's carbon-dioxide level rises. Spinal reflex respiratory-like movements may occur despite brainstem failure and may, along with hyperdynamic cardiac contraction, trigger the patient's ventilator if the sensitivity is set too low, giving the false impression of spontaneous breathing efforts. Such movements, however, typically occur late in the test as a result of acidosis and/or hypoxemia, do not result in significant tidal volumes, and will show no change in the

patient's end-tidal carbon-dioxide waveform unless they are of sufficient strength to trigger the patient's ventilator.

When appropriate, a 10 minute apnea test is performed according to the "Apnea Test Procedure" following pre-oxygenation for at least 10 minutes with a FiO_2 of 1.0 and normalization of the patient's $PaCO_2$ to 40 mmHg. The certifying physician must be present throughout this study to document the presence of apnea as well as to be available to intervene should the patient became hemodymically unstable during test. Patients with an intact brainstem can be expected to breathe within the first few minutes of the test as hypercarbia typically develops at a rate of 3 mmHg per minute. Spontaneous body movements are generated by the spine, typically in response to physical stimulation. As a result, the patient should not be stimulated in any way during the apnea trial to avoid the development of spontaneous movements that might interfere with accurate interpretation of the test's results.

A patient is considered to meet apnea test criteria for brain death if:

1) No spontaneous respiratory efforts were witnessed during the test (as evidenced by physical attempts to inspire or documentation of end-tidal carbon dixoide by bedside waveform analysis) AND

2) The patient's $PaCO_2$ is in excess of 60 mnHg (or at least 20 mmHg above baseline)

In the presence of these two findings, and in conjunction with an appropriate clinical examination, the patient meets accepted criteria for brain death and such documentation is made in the patient's medical record. Once two independent determinations of brain death have been made and each physician has documented their findings and opinion in the medical record, discontinuation of mechanical ventilator support may occur.

CONFIRMATORY LABORATORY TESTS

A confirmatory test is not mandatory, but is desirable in patients in whom specific components of clinical testing cannot be reliably performed or evaluated. The following confirmatory test findings are listed in order of the most sensitive test first.

1. Conventional angiography

   No intracerebral filling at the level of the carotid bifurcation or circle of Willis. The external carotid circulation is patient, and filling of the superior longitudinal sinus may be delayed.

2. Electroencephalography

   No electrical activity during at least 30 minutes of recording that adheres to the minimal technical criteria for EEG recording in suspected brain death

3. Transcranial Doppler ultrasonography

   Small systolic peaks in early systole without diastolic flow or reverberating flow, indicating very high vascular resistance associated with greatly increased intracranial pressure. Ten percent of patients may not have temporal insonation windows precluding use of this technique for determining brain death.

4. Technetium- 99mm cerebral blood flow scans

   No uptake of isotope in brain parenchyma (“hollow skull phenomenon”)

5. Somatosensory evoked potentials
   Bilateral absence of N 20-P22 response with median nerve stimulation

CLINICAL CONDITIONS THAT MAY INTERFERE WITH THE DIAGNOSIS OF BRAIN DEATH

The following physical conditions may interfere with the clinical diagnosis of brain death.[3] In such situations, confirmatory tests are recommended as clinical neurologic examination alone may not be accurate.

- Severe facial trauma
- Preexisting papillary abnormalities
- Toxic levels of any sedative drugs, aminoglycosides, tricyclic antidepressants, anticholinergics, antiepileptic drugs, chemotherapeutic agents, or neuromuscular blocking agents.
- Sleep apnea or severe pulmonary disease resulting in chronic retention of carbon dioxide ($PaCO_2$)

CLINICAL OBSERVATIONS COMPATIBLE WITH THE DIAGNOSIS OF BRAIN DEATH

The following physical findings are occasionally seen and should not be misinterpreted as evidence for brainstem function:

- Spontaneous limb movements other than pathologic flexion or extension response
- Respiratory-like movements (shoulder elevation and adduction, back arching, intercostals expansion without significant tidal volumes)
- Sweating, blushing, tachycardia
- Normal blood pressure without pharmacologic support or sudden increase in blood pressure
- Absence of diabetes insipidus
- Deep tendon reflexes; superficial abdominal reflexes; triple flexion response
- Babinski reflex

MEDICAL RECORD DOCUMENTATION

Following the determination of brain death, appropriate documentation in the patient's medical record should be performed[3]. This documentation should include the following components:

- Etiology and irreversibility of the patient's coma and overall clinical condition.
- Absence of brainstem reflexes
- Absence of motor response to pain
- Absence of spontaneous respiration despite a $PaCO_2 \geq 60$ mmHg (when apnea testing can be safely or appropriately performed)
- Justification for and result of additional confirmatory test
- Findings or repeat neurologic examination (typically performed by a second physician)

Management of Central Nervous System Infections

ICU QUALITY ASSURANCE PROGRAM

1. **DOCUMENTED SYSTEM**
   a. The policy and procedures to be followed are laid down in the form of ICU manual.
   b. ICU staffs are to follow the laid down procedures.
2. **MONITORING**
   a. Medical Administration department and In charge of ICUs are responsible to ensure and monitor compliance to laid down procedures. Internal Quality Audit to be conducted by Quality Assurance Department with identified technical experts.
   b. Daily rounds by Consultants, RMO, Administrator and Manager Operations ensures monitoring on day to day basis
3. **PERFORMANCE REVIEW**
   Medical Administration department and In charges of ICUs to review the performance of ICU, which includes the following

a. ALOS
b. Bed Occupancy Rate
c. Death Rate
d. Care of environment (Fumigation, Culture reports)
e. LAMA
f. HAI
g. Patient attendant satisfaction level
h. Bed turnover rate (BTR)
i. Bed Shortage Incidences
j. Counselling (Patient Education)
k. Sentinel Events
l. Barrier Nursing Compliance
m. Near Misses
n. Hand washing compliance
o. Patient and Family Education
p. Compliance to patient safety goals
q. VAP
r. CRBSI
s. Readmissions
t. Reintubations
u. Surgical wound infection rate
v. Door to Ballon time for PCI, Bleeding rate after angiogram (CICU).

4. **CORRECTIVE / PREVENTIVE ACTION**

4.1 To identify the gaps through monitoring system (Internal Quality Audit, Daily Rounds and Performance Review).

4.2 To find the Root Cause(s) and take appropriate corrective / preventive action (s).

i. **FOLLOW UP**

All corrective / preventive action(s) to be evaluated for its effectiveness.

ii. **TRAINING**

a. Respective In charges of ICU to identify the training needs and coordinate for the same.

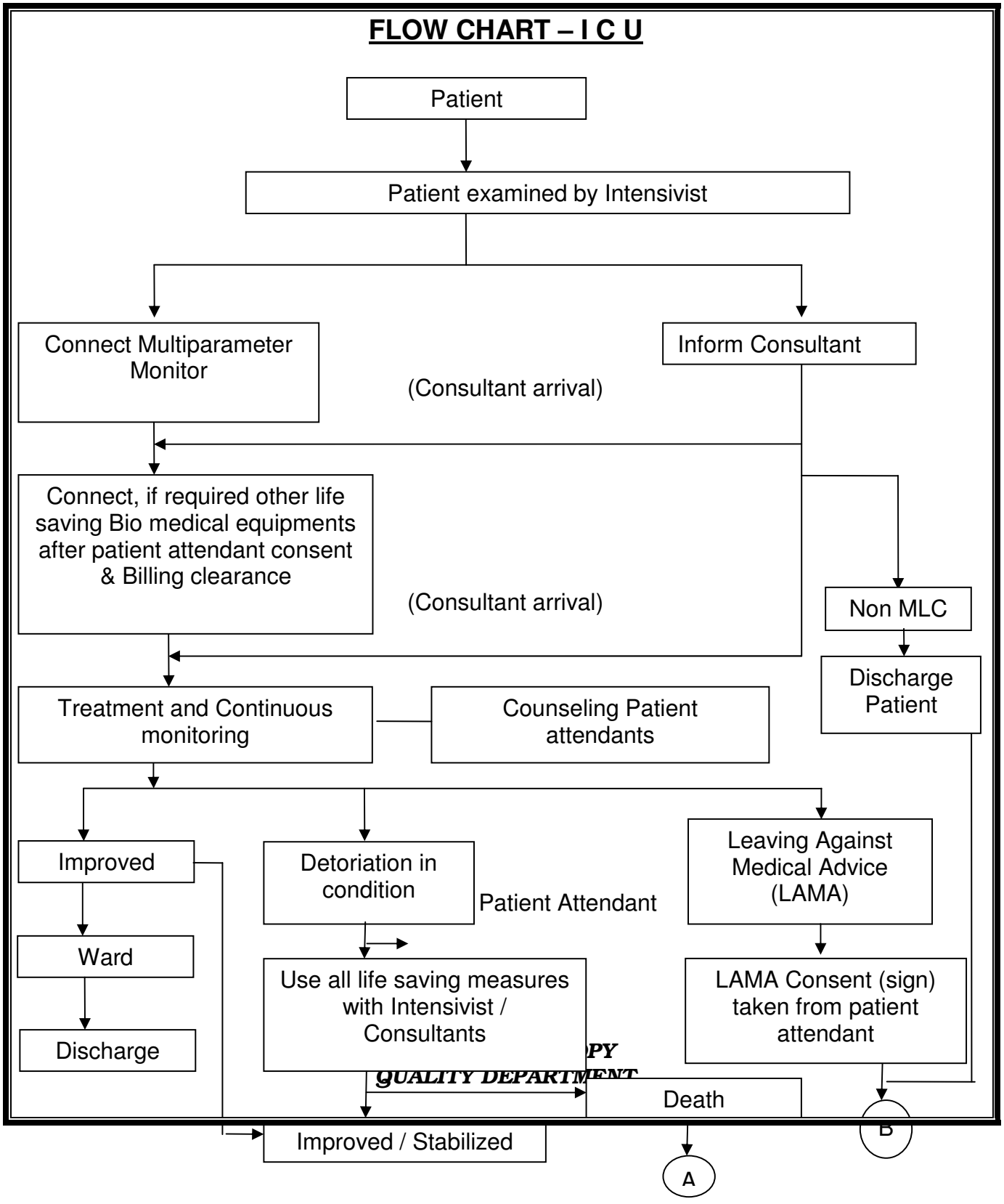
b. Periodical training to be conducted for ICU staff.

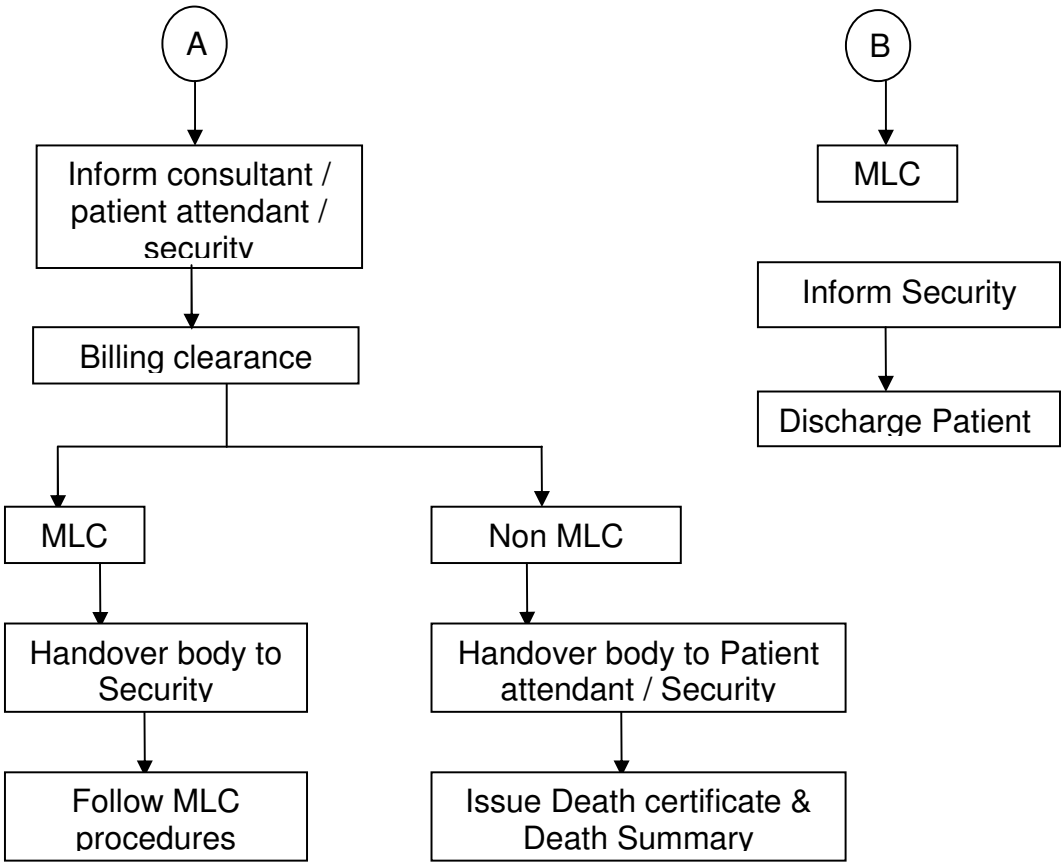
c. Twice a year training to be given to the ICU staff on the documented system (ICU Manual).

iii. **CONTINUAL IMPROVEMENT**

In charge ICU to identify areas for continual improvement. Quality Improvement plan to be made for the identified areas. The effectiveness of implementation to be monitored. Quality Assurance Department to support In charge ICU for the same.

FLOW CHART – I C U




A
Inform consultant / patient attendant / security
Billing clearance
MLC
Handover body to Security
Follow MLC procedures
Non MLC
Handover body to Patient attendant / Security
Issue Death certificate & Death Summary
B
MLC
Inform Security
Discharge Patient

ABBREVIATIONS.

1. HONK – Hyperosmolar Hyperglycaemic Non-Ketotic Coma.
2. SjO_2 – Jugular Bulb Venous Oxygen Saturation
3. CVC – Central Venous Catheter
4. HITS – Heparin induced thrombocytopenia
5. PAC – Pulmonary Artery Catheter
6. SVC – Superior vena cava
7. RA – Rheumatoid Arthritis
8. SVR – Systemic Vascular Resistance
9. RAP – Right Arterial Pressure
10. PAOP – Pulmonary Artery Occlusion Pressure
11. PAP – Pulmonary Arterial Pressure
12. VO_2, DO_2 –Venous oxygen consumption, Oxygen delivery /supply.
13. UWSD – Under water sealed drain system
14. LVSWI – Left Ventricular Stroke Work Index
15. CO/CI – Cardiac output/Cardiac input
16. PVR – Pulmonary Vascular Resistance
17. LMA – Laryngeal Mast Airway
18. CP – Cricoid Pressure
19. FiO2 – Fraction of inspired oxygen
20. EVAC – Endotracheal tube with evacuation
21. FVC – Forced Vital Capacity

22. BAL – Broncho Alveolar Lavage
23. IABP – Inter aortic balloon pump
24. PTCA – Percutaneous Transluminal Coronary Angioplasty
25. CAVG – Cardiac Access Vascular Graft
26. VSD – Ventricular Septal Defect
27. AMI – Acute Mitral Incompetence
28. ECM – External Cardiac Message
29. TVP – Transvenous pacing
30. CAO – Chronic Airflow Obstruction
31. TPN – Total parental Nutrition
32. PEG – Percutaneous endoscopic gastrostomy
33. PEJ - Percutaneous endoscopic jejunostomies
34. PICC – Peripherally inserted central catheter
35. SIADH – Syndrome of inappropriate antidiuretic hormone
36. ECFV – Extra cellular fluid volume
37. ADH – Anti diuretic hormone
38. DDAVP – Desmopressin acetate 1-deamino-8-d-arginine Vasopressin
39. DI – Diabetes Insipidus
40. JGA – Juxtaglomerular apparatus
41. GFR – Glomerular Filteration rate
42. PIOPED – Prospective investigation of pulmonary diagnosis
43. DFAT – Direct Fluorescent antibody test
44. ELISA – Enzyme linked immunoassay
45. GBS – Guillain Barre Syndrome
46. SBH – Subarachnoid hemorrhage
47. DBP – Dibutyl Phthalate

48. STK – Streptokinase